DOES NOT CIRCULATE

FOUNDED IN 1906 AS THE AMERICAN QUARTERLY OF ROENTGENOLOGY

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

UNIVERSITY OF MICHIGAN
SEP 29 1955
MEDICAL

OFFICIAL ORGAN OF
THE AMERICAN ROENTGEN RAY SOCIETY
THE AMERICAN RADIUM SOCIETY

LAWRENCE REYNOLDS, Editor

ASSOCIATE EDITORS

HARRY HAUSER TRAIAN LEUCUTIA RUSSELL H. MORGAN
EDWARD B. D. NEUHAUSER WENDELL G. SCOTT

CONSULTING EDITORIAL BOARD

PAUL C. AEBERSOLD
OSCAR V. BATSON
JOHN CAFFEY
JAMES T. CASE
ARTHUR C. CHRISTIE
ROSS GOLDEN
PAUL C. HODGES
HOWARD B. HUNT
B. R. KIRKLIN
JOHN H. LAWRENCE
REED M. NESBIT
EUGENE P. PENDERGRASS
U. V. PORTMANN
MERRILL C. SOSMAN
LAURISTON S. TAYLOR
OWEN H. WANGENSTEEN
SHIELDS WARREN

Issued Monthly for the American Roentgen Ray Society by
CHARLES C THOMAS · PUBLISHER · SPRINGFIELD · ILLINOIS

VOLUME 74 SEPTEMBER, 1955 NUMBER 3

Fifty-sixth Annual Meeting, American Roentgen Ray Society
Palmer House, Chicago, Illinois, September 20-23, 1955

For Program see July issue; for Instruction Courses see August issue

WESTINGHOUSE ANNOUNCES THE

AEROFLEX

An overhead tubestand with tremendous vertical, transverse and longitudinal coverage.

See it at the fifty-sixth annual meeting of the American Roentgen Ray Society —Palmer House, Chicago, September 20, 21, 22 and 23, 1955.

YOU CAN BE SURE... IF IT'S

Westinghouse

MEDICAL X-RAY

J-08312

Westinghouse Electric Corporation

X-RAY DIVISIO

2519 Wilkens A

Baltimore 3, Ma

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 74 SEPTEMBER, 1955 No. 3

CONTENTS

Pathology of Carcinoma of the Lung as Related to the Roentgen Shadow. AVERILL A. LIEBOW, M.D. 383

The Detection of Carcinoma of the Lung by Screening Procedures, Particularly Photofluorography. L. H. GARLAND, M.B. 402

The Roentgen Signs of Carcinoma of the Lung. LEO G. RIGLER, M.D.. . . 415

Surgical Procedures in the Diagnosis and Treatment of Bronchogenic Carcinoma. EDGAR W. DAVIS, M.D., SOL KATZ, M.D., AND J. WINTHROP PEABODY, JR., M.D. 429

Pulmonary Arteriovenous Fistulas. WALTER J. STORK, M.D. 441

Sarcoidosis; An Analysis of Twenty-One Proved Cases. ALVIN W. FINESTONE, M.D. 455

Roentgenological Observations on Pleuropulmonary Tularemia. JOSEPH McK. IVIE, M.D. 466

Combined Hiatus Hernia and Jejunal Diverticulum; Report of a Case and Review of the Literature. EDMUND W. KLINEFELTER, M.D. 472

Roentgen Diagnosis of Esophageal Varices; Comparison of Roentgen and Esophagoscopic Findings in 502 Cases. I. E. KIRSH, M.D., C. C. BLACKWELL, M.D., AND H. D. BENNETT, M.D. 477

Gas in the Portal Veins of the Liver in Infants; A Roentgenographic Demonstration with Postmortem Anatomical Correlation. JOHN N. WOLFE, M.D., AND WILLIAM A. EVANS, M.D. 486

Postcaval Ureter; A Case Report. D. A. VAN VELZER, M.D., C. W. BARRICK, M.D., AND E. L. JENKINSON, M.D. 490

Studies on Nagasaki (Japan) Children Exposed In Utero *to the Atomic Bomb.* WATARU W. SUTOW, M.D., AND EMORY WEST, M.D. . . . 493

Radium Therapy in Cancer of the Skin and Lip. B. L. FEUERSTEIN, M.D. 500

(Contents continued on p. iv)

Experimental Orbitography. P. Thomas Manchester, Jr., M.D., Jose Bonmati, M.D., Ted F. Leigh, M.D., and F. Phinizy Calhoun, Jr., M.D. 508

Roentgen-Ray and Electron Radiography of Thin Specimens. Arthur I. Berman and James W. Dutli 518

Editorials

Fibrous Defects in the Cortex of Growing Bones. Frederic N. Silverman, M.D. 527

William H. Van Strander, 1874–1955. Wendell C. Hall, M.D. . 529

Louis Edeiken, 1894–1955. J. Gershon-Cohen, M.D. 530

Society Proceedings, Correspondence and News Items 532

Abstracts of Radiological Literature 538

CONSOLIDATED INDICES

Volume I (1903-1937) $12.50
Volume II (1938-1942) 5.00
Volume III (1943-1947) 12.50
Volume IV (1948-1952) In Process

Order From:
CHARLES C THOMAS · PUBLISHER
301-327 East Lawrence Avenue
Springfield, Illinois

(Purchasers outside U.S.A. should send cash with order. Delivery, postpaid, guaranteed.)

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 74 SEPTEMBER, 1955 No. 3

PATHOLOGY OF CARCINOMA OF THE LUNG AS RELATED TO THE ROENTGEN SHADOW*

By AVERILL A. LIEBOW, M.D.
NEW HAVEN, CONNECTICUT

A DETAILED consideration of the histopathology and histogenesis of cancer of the lung cannot be given here; rather the attempt will be to reconcile shadow with substance, stressing knowledge recently acquired, or in the gathering.

SOME GENERAL CONSIDERATIONS

Recent angiographic studies have focussed interest on changes in the intrathoracic vessels produced by tumors of the lung.[4,23] Some of these are obvious, such as evidence of displacement, gross irregularity, or even obstruction of large intrathoracic veins with establishment of a collateral circulation. Involvement of main pulmonary arteries may be similarly demonstrated. Less widely understood, however, is the fact that when lung tissue is rendered immobile it receives less blood. This is consequent to the very important influence of the bellows action of the thorax in pumping blood. When this action is interrupted on one side, blood, and with it the angiographic contrast medium, is directed into the uninvolved side. In time the vessels on the diseased side tend to become smaller. With reference to the intimate blood supply of the tumors, it has now been well established that primary tumors receive their blood from the systemic bronchial arterial system.[3,13,29] Pulmonary arteries and veins, on the other hand, may actually be displaced by the tumor at its periphery (Fig. 1). This might under favorable circumstances be demonstrable roentgenographically, as, for example, by catheterization and segmental angiography. This procedure will not, however, distinguish neoplasm from other lesions that also receive blood from the aorta rather than the pulmonary vessels, *e.g.*, tubercles, or bronchogenic cysts. It may happen that pre-existing pulmonary arteries and veins become surrounded by the growing neoplasm although not obstructed, but this does not necessarily imply that they receive blood from these vessels. Perhaps this accounts for the diverse statements that have been made regarding the blood supply of pulmonary neoplasms. On the contrary, as some have stated, it may be that metastatic tumors receive their blood supply from the pulmonary arteries.[3]

* From the Department of Pathology, Yale University School of Medicine, New Haven, Connecticut. Presented in a Symposium on Carcinoma of the Lung at the Fifty-fifth Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

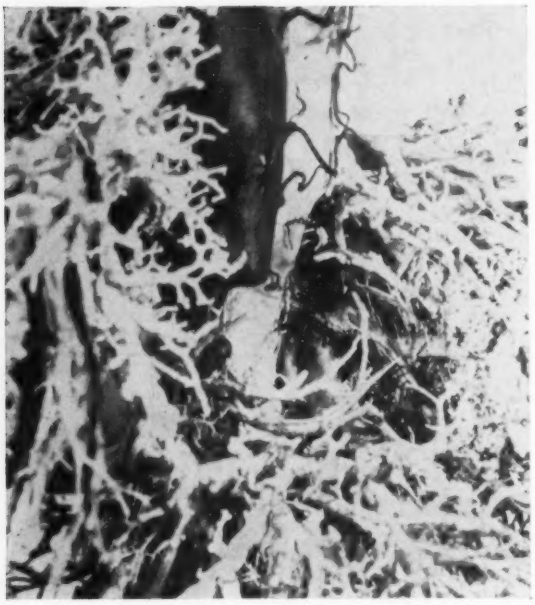

Fig. 1. Blood supply of epidermoid carcinoma. Vinylite cast. A spherical mass has compressed the intermediate bronchus of the right lung into a thin ribbon and displaced the pulmonary arteries and veins, which are likewise flattened and distorted. The true blood supply of the tumor is derived from greatly enlarged bronchial arteries (black), best seen in the region of the former capsule. (Reproduced by permission from Liebow.[13])

In our still limited experience this has not been confirmed and they have not differed from the primary lesions in their vascularization from the systemic bronchial arteries (Fig. 2).

Less than the needed intensive study has been given to the lymphatic drainage of the lung in man. Observations of Rouvière[22] and others suggest that the superior portion of the left upper lobe drains to lymph nodes of the left side: the left paratracheal group, the node of the ductus arteriosus, and the left anteromediastinal group including the node of the recurrent nerve. From other portions of the left lung, including the inferior portion of the upper lobe, drainage is in part into the subcarinal group; thence it may extend toward the right side of the mediastinum. On the right side, drainage from the middle and inferior lobes is into the subcarinal lymph nodes, as well as other portions of the mediastinum, especially of the right side. Techniques have been described for removing these masses of lymph nodes including the subcarinal group in a single block with the appropriate lung.[1,28] The value of this "radical pneumonectomy" in prolonging life remains to be determined. It is obvious that the plane of dissection, even under the best circumstances, must pass within a millimeter of any tumor-bearing lymphatics or nodes. The mere presence of enlarged lymph nodes in association with bronchogenic carcinoma does not necessarily indicate the presence of metastatic tumor. These lymph nodes may be the seat of lymphadenitis consequent to a pneumonitic process. Their roentgenographic demonstration by laminagraphic techniques or in esophagograms should therefore not be interpreted as an inevitable sign of a bad prognosis.

Fig. 2. Blood supply of pulmonary metastasis from hypernephroid carcinoma. Vinylite cast. Two small pulmonary arterial branches (*1* and *2*) join at an obtuse angle, since they have been displaced by a nodule of tumor. A skeleton of bronchial arteries remains after the tumor tissue has been digested away. The feeders of these vessels are seen extending from the hilus below and to the right along the bronchi leading to the involved parenchyma.

CLASSIFICATION

A study of many malignant tumors of the lung has demonstrated that their natural history depends in some measure upon their microscopic structure. Since different parts of the same bronchogenic carcinoma may vary microscopically, the classification is made according to the predominant cell type and presumed histogenesis, but the term "mixed" is applied when this is not possible. Malignant epithelial tumors can accordingly be classified as:

A. Bronchogenic carcinoma
   1. Epidermoid carcinoma
   2. Anaplastic carcinoma
   3. Adenocarcinoma
   4. Mixed type

B. Bronchiolar carcinoma ("Pulmonary adenomatosis," "Alveolar cell carcinoma," etc.)

[C. "Bronchial adenoma"]
   1. Carcinoid type
   2. Cylindromatous or adenocystic type
   3. Others

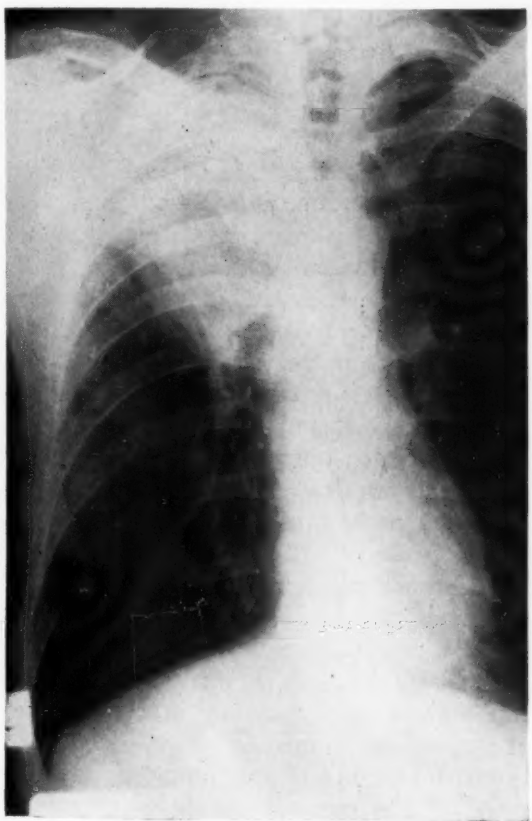

FIG. 3. Epidermoid carcinoma. A rather homogeneous shadow occupies the upper portion of the right lung field. Compare with Figure 4. (Reproduced by permission from Lindskog and Liebow.[14])

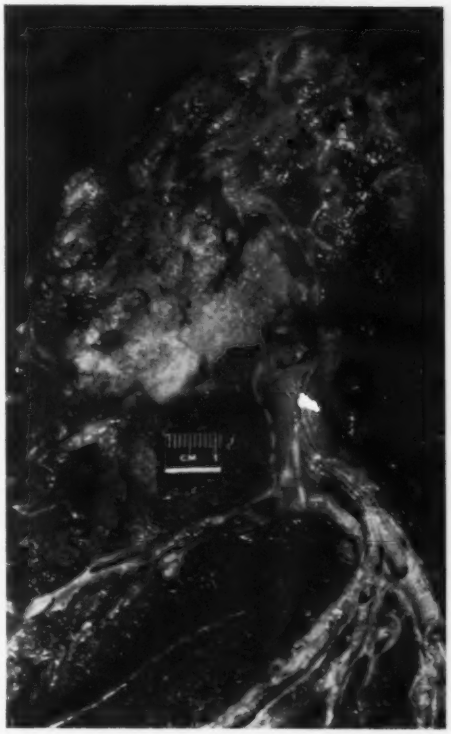

FIG. 4. Epidermoid carcinoma. After transection of the resected lung, it is obvious that the roentgen shadow represents the entire right upper lobe and was cast only in part by tumor; but in part also by atelectatic tissue which is also the seat of an organizing pneumonitis. The tumor itself is pear shaped, with its bulk in the anterior segment, and a relatively narrow part that extends proximally to obstruct the upper lobe bronchus. The unobstructed middle lobe bronchus is seen below the metric scale. Separated by the still complete fissure is the lower lobe. (Reproduced by permission from Lindskog and Liebow.[14])

Although this classification is not universally accepted, it has the merit of simplicity. As simple a classification as possible should be retained until specific subgroups can be delineated. Opinions differ as to whether the "bronchial adenomas" should be classified with the other tumors in this group. They are therefore bracketed and the problem will be dis-

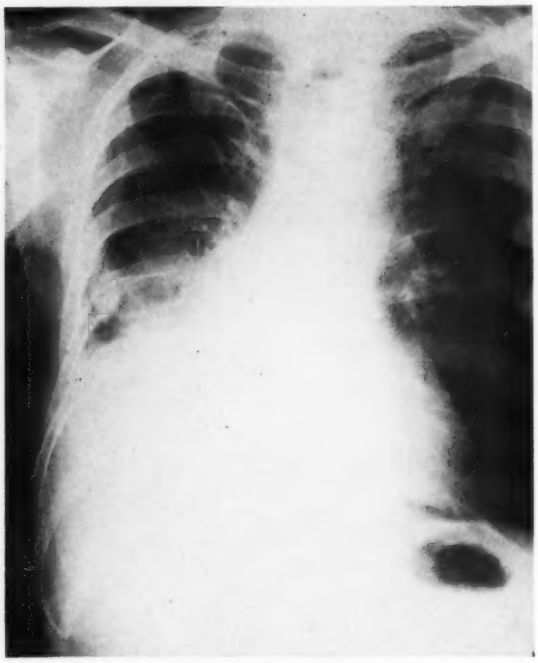

Fig. 5. Epidermoid carcinoma. Shadow in the lower lung field interpreted as representing largely tumor. Irradiation was carried out over a seven month period, and the right lung was then resected. Compare with Figure 6.

cussed briefly. The rare carcinosarcomas will be considered beyond the scope of this brief survey.

Epidermoid Carcinoma. Epidermoid carcinoma tends to predominate by far in the male sex (in a ratio variously quoted as 10:1 up to 20:1). The peak incidence is close to age sixty, later than that of other bronchogenic tumors.[2,18]

Epidermoid carcinoma comprises between 45 and 60 per cent of all bronchogenic tumors, depending upon what is included in the group. Transitions exist from tumors composed of keratinized squamous epithelium containing epithelial pearls and clearly demonstrable intercellular bridges, and forms undifferentiated, but still recognizable as epidermoid tumors analogous to those of the skin. Where the line of distinction is drawn between the latter and carcinomas classified as anaplastic is arbitrary. This explains the differences in statements regarding the natural history of these tumors.[2,23,15] A small proportion of tumors classified here as epidermoid, but put by others in a separate group,[24] consists of remarkably polymorphous elements with giant cells and spindle forms, that may make distinction from sarcoma difficult. Actually these seem to behave as relatively slowly growing epidermoid tumors with a marked tendency to cavitation. Their epidermoid nature may be apparent only in certain fields.

Grossly epidermoid carcinoma appears most commonly as a pear-shaped mass of translucent pink tissue flecked with the black of anthracotic material and with the chalky yellow of foci of necrosis. It surrounds in bulk a lobar or segmental bronchus, and frequently one or several orders of branches beyond. Since the pulmonary tissue is likely to have suffered alteration consequent to obstruction of the bronchus, it is usually not possible to state how much of the roentgen shadow is actually tumor. Since the narrower end of the "pear" is closer to the hilus it appears probable that the actual point of origin of most tumors is beyond the bronchus most proximally involved (Fig. 3 and 4). A stalk of tumor may actually present within the lumen of the bronchus at the hilar end of the lesion. This can sometimes be demonstrated roentgenographically, particularly by the sectioning techniques, as an abrupt interruption of the bronchus. Proximal extension may also be demonstrated microscopically within the wall of the bronchus deep to the surface, but usually not more than 1 or 2 cm. proximally of the grossly visible lesion.[9] The tumor may, however, reappear on the surface proximal to the main mass, with an irregular ring of uninvolved mucous membrane between.

Rather than expanding into tissue surrounding the bronchus of origin, epidermoid tumors may remain more or less confined to the lamina propria, with the replacement of the normal epithelium by irregular masses of tumor cells, and the lumen of the bronchus may be minimally narrowed or not at all. Bronchial drainage

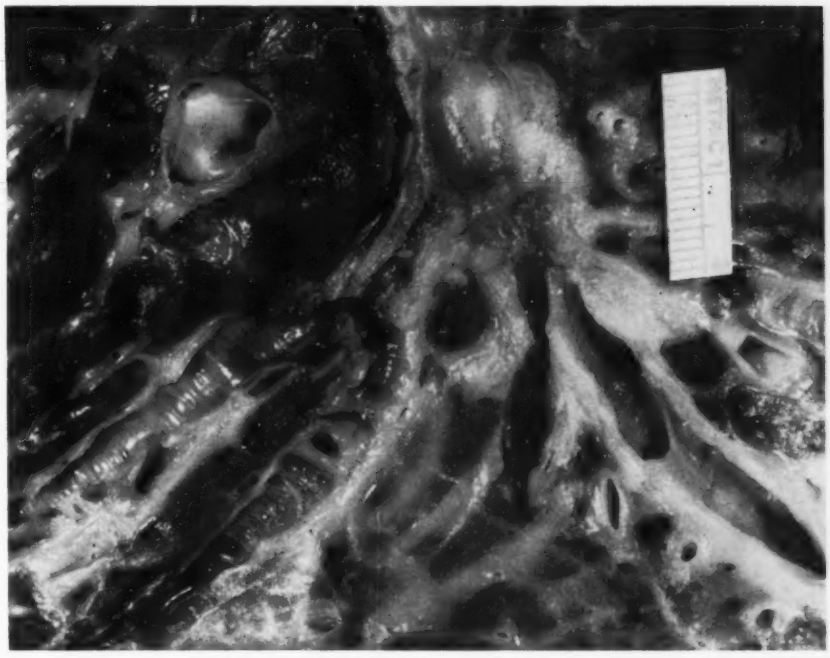

Fig. 6. Epidermoid carcinoma. The actual tumor is very small and presents a rough elevation of the surface of the bronchus. It has narrowed the intermediate bronchus, and extends for a short distance into the middle and right lower lobe branches. The tumor extends to the lymph node between these major branches. The middle lobe bronchus extends to the right and above the lymph node, and a narrowed lower lobe bronchus downwards and to the left.

may nevertheless be disturbed by a relatively small lesion of this type, the bronchoscopic appearance of which often suggests granulation tissue rather than neoplasm. Such a tumor no less than a truly obstructing mass may have behind it a large zone of pneumonitis or its complications such as bronchiectasis or abscess. Thus the size of the roentgen shadow is not necessarily a guide to the size of the actual neoplasm (Fig. 5 and 6). There have been unfortunate instances of delay in surgical treatment, occasioned by attempts to "shrink" with radiation therapy such a presumed tumor mass, which in reality represents consolidated but not carcinomatous tissue.

Occasionally a bizarre distribution of the lesion may be encountered roentgenographically, when the tumor invades a somewhat remote bronchus by penetrating from a lymph node. Thus there may be an irregular mass in one lobe, and a wedge-shaped zone of atelectasis, or pneumonitis in another (Fig. 7–9).

The epidermoid tumors are not confined to the major bronchi, and may present as peripheral, or even subpleural, masses. Such lesions tend to be relatively silent clinically until the pleura or body wall is invaded. A lung tumor that invades the body wall is more likely to be a very well differentiated squamous cell tumor than any other type of bronchogenic carcinoma (Fig. 10 and 11). Perhaps the reason for this is that adenocarcinoma, although more commonly peripheral in location, is likely to have metastasized and to have manifested itself in consequence of this before the body wall has become invaded. When such a well differentiated epidermoid tumor takes origin in the periphery of the upper parts of the lung, as for example in a tuberculous cavity, invasion of structures at the upper thoracic inlet may take place. This produces a characteristic group of symptoms described by Pancoast, the superior sulcus or Pancoast syndrome,[11,16] in which there is usually destruction of the

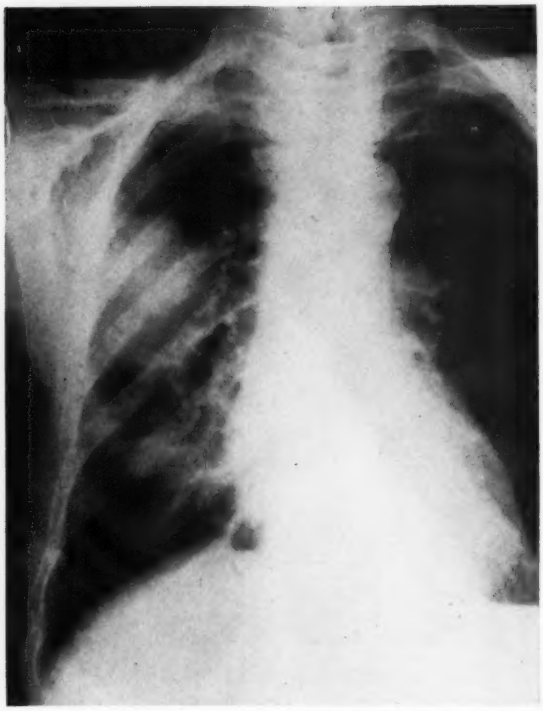

Fig. 7. Epidermoid carcinoma. Rounded density in the right upper lobe. A wedge-shaped zone representing atelectasis of the middle lobe with its base upon the hilus, extends part way into the lung field. Compare with Figures 8 and 9.

sympathetic ganglions with Horner's syndrome, loss of power in the arm, pain, edema of the upper extremities and evidence of destruction of ribs or vertebrae. A similar syndrome, of course, may result from any destructive lesion in this portion of the body, but statistically it is predominantly the consequence of well differentiated squamous cell carcinoma.

Necrosis commonly occurs in epidermoid tumors, especially in the more differentiated squamous forms, and frequently there is discharge of necrotic material with cavitation. Cavities reach a size in excess of 4 cm. in diameter in approximately 20 per cent of epidermoid tumors by the time they are seen at necropsy. Occasionally the cavitation far exceeds this magnitude (Fig. 10 and 11). Thus carcinoma may have a roentgenographic appearance suggesting abscess. Such lesions, however, rarely have a history or symptomatology appropriate to true abscess, and they may occur anywhere in the lung. Their walls tend to be relatively thick and are sometimes demonstrably irregular. The differential diagnosis can be difficult when the lining is apparently smooth, and when the lesion is located in a part of the lung where abscess is common, *i.e.*, in the "path of aspiration": the posterior segments of the upper lobe, and the superior and basal segments of the lower lobe (Fig. 12 and 13). Nor do all pyogenic abscesses have a typical history and symptomatology. Expert cytological examination of sputum or bronchial secretions is important in the differential diagnosis, when bronchoscopic or other techniques have failed.[14] It is remarkable that even the most thin walled of these cavita-

Fig. 8. Epidermoid carcinoma. The posterior position of the mass shown in Figure 7 is demonstrated, as well as the densely atelectatic middle lobe. This film is printed in reverse of the usual, for comparison with Figure 9.

tions associated with bronchogenic carcinoma are usually lined by an uninterrupted layer of tumor cells.[13]

It is of course possible that a cavitative lesion may consist in part of necrotic tumor, and in part of necrotic lung. This is sometimes demonstrably the consequence of invasion of blood vessels by the tumor with infarction of the lung. Under these circumstances bacterial infection often is superimposed and becomes a complicating and clinically confusing factor. True abscess behind an obstructing carcinoma of the lung is relatively uncommon. When the lesion is in the apical or posterior segments of the lung, cavitative tuberculosis may be closely simulated.

It seems probable from recent observations on patients who have been "followed" after a questionable roentgen shadow has been demonstrated, and from the valuable review by Rigler and his coworkers[20] of early survey films of patients who later proved to have neoplasms, that some bronchogenic carcinomas, especially of the epidermoid type, may be more slowly growing and have a much longer existence before identifying symptoms appear than has been realized in the past (Fig. 14–17). Their course, once the diagnosis is made, is a very variable one.

Fig. 9. Epidermoid carcinoma. The primary tumor is seen to occupy almost the entire posterior segment of the right upper lobe. Tumor from a lymph node (arrow) has invaded the middle lobe bronchus, producing a high degree of stenosis. The entire middle lobe is atelectatic and the seat of "obstructive pneumonitis." Its pale appearance contrasts with the darker color of the overexpanded anterior segment that lies above it. Below the origin of anterior segmental bronchus is another mass of lymph nodes that has not significantly compressed it.

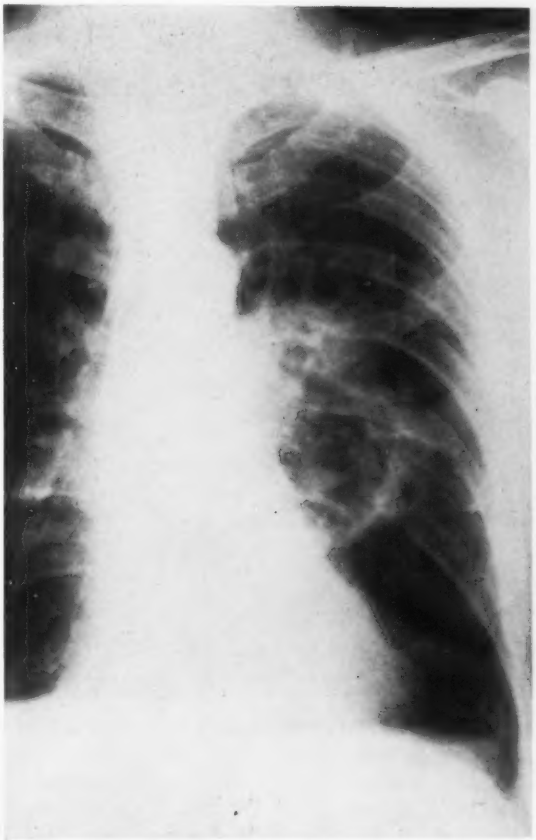

Fig. 10. Epidermoid carcinoma. The region of the superior segment of the left upper lobe is occupied by an obviously cavitated mass. In the upper third of this cavity there is a roughly triangular density with its base upon the hilus, and another, shelf-like, protrusion is seen below it. Compare with Figure 11.

Epidermoid carcinoma may reach an enormous bulk by continuous growth when situated in the center of a lobe, without invasion of surrounding structures, nor extrathoracic metastases and even without alarming symptoms of pulmonary dis-

Fig. 11. Epidermoid carcinoma. The lateral half of the lung shown in Figure 10 viewed from the medial side after resection. Not only the superior segmental bronchus, but also the posterior basal bronchus, is in communication with the huge cavitated mass The triangular density noted in Figure 10 is seen to be tumor tissue lipping the superior segmental bronchus as it enters the cavity. The shelf-like protrusion below is a similar mass above the communication of the posterior basal bronchus. Posteriorly (at the left of the photograph) there is dense white tissue representing pleura and endothoracic fascia invaded by the well differentiated squamous tumor.

ease (Fig. 17). Growth along bronchial walls has already been discussed. One feature of the anatomy of the lung that creates a problem in the surgical treatment of carcinoma is the intimate relation of certain bronchi to the pulmonary ligament and mediastinum. The dissection as ordinarily carried out then passes through a thin sheet of areolar tissue, hopefully separating the tumor with the lung, from the structures left behind. When such bronchi are involved an intrapericardial approach to the vessels during pneumonectomy would seem to afford a better chance of cure.

More so than malignant bronchogenic tumors of other types, the epidermoid carcinomas, and especially those to which the designation squamous is applicable, seem for a long time to remain confined to the parenchyma and to lymph nodes immediately adjacent to the bronchus of origin (Fig. 4 and 6). In fact, such nodes seem at first to be engulfed by the primary mass rather than entered by lymphatic permeation. True lymphatic extension ultimately occurs (Fig. 9).

Involvement of lymph nodes has a definite prognostic significance, as indicated by the observations of Carlisle, McDonald and Harrington;[2] 63.2 per cent of patients survived five years after resection when the regional lymph nodes showed no evidence of carcinoma in routine microscopic examination, whereas in those whose nodes contained tumor the five year survival rate was only 25 per cent.

With the growth of the tumor, pulmo-

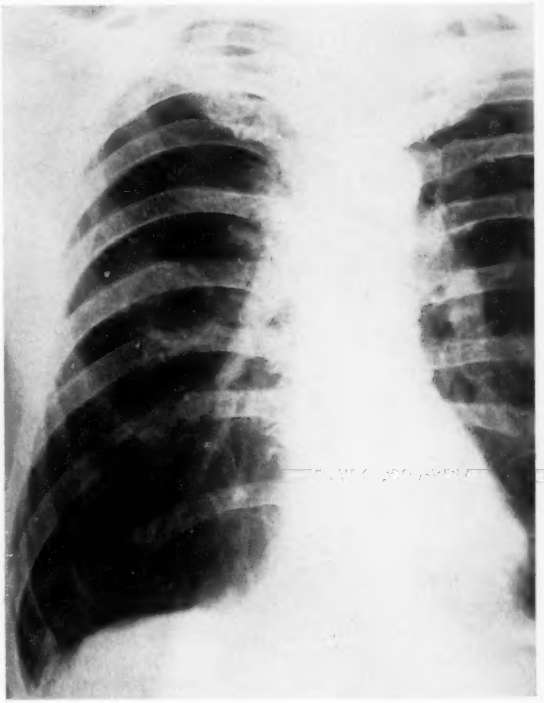

Fig. 12. Epidermoid carcinoma. A thin-walled cavity is seen in the mid-portion of the right lung. Lateral views demonstrated this cavity to be in the superior segment of the lower lobe. Compare with Figure 13.

nary arteries and veins may be invaded rather than merely displaced. Invasion of arteries usually does relatively little harm, but in some instances there may be infarction of the lung beyond. Invasion of pulmonary veins opens the entire systemic circulation to metastasis. Expanded collateral bronchial venous channels connect with the azygos, and thus the spinovertebral veins—a pathway to the bony axis and central nervous system.

Although hematogenous metastases tend to appear relatively late in epidermoid carcinoma they nevertheless pursue the same general pathways as other types of bronchogenic carcinoma. Most commonly involved at the time of necropsy, besides the regional lymph nodes, are the liver, bones, adrenals, and kidneys. Less frequent is metastasis to the brain, although evidence of neurological disturbance may be the first sign of the disease. The pericardium and heart are occasionally involved, chiefly by direct extension, and the pancreas may be the seat of lymphogenous metastasis.

Fig. 13. Epidermoid carcinoma. Medial aspect of the lung shown in Figure 12. The cavity has a rather smooth lining with only a few rounded irregularities. This appearance might suggest abscess, but is belied by the extensive anthracotic pigmentation that is more indicative of tumor. The posterior wall is thicker than would be suggested by the roentgenographic appearance as seen in Figure 12.

Fig. 14. Epidermoid carcinoma. Rounded mass visualized in November, 1950, when the patient, a hospital employee, was sixty-nine years old. This is a routine film in an essentially asymptomatic individual.

Anaplastic Carcinoma. Relatively more of the anaplastic carcinomas occur below the age of forty than is the case with epidermoid tumors, although the peak incidence is only slightly lower. The predominance in the male sex also is somewhat greater. Approximately 30 per cent of the bronchogenic tumors in the present classification fall into the anaplastic group.

Among anaplastic tumors belong the

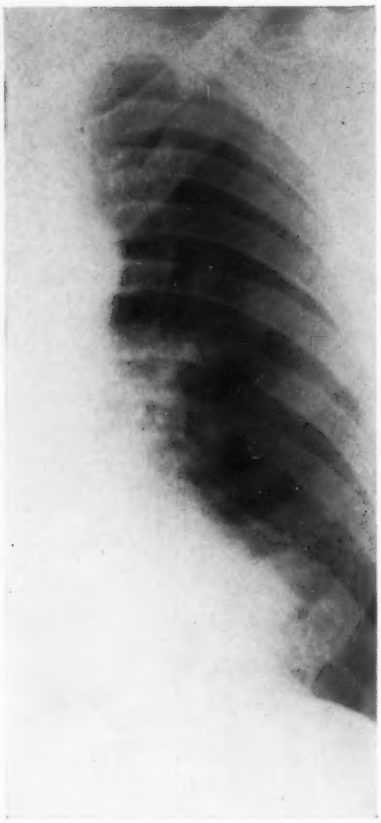

Fig. 15. Same patient as in Figure 14. Film of January, 1952. Progression in size of the nodule is demonstrated.

"oat cell" carcinomas,[15] called "reserve cell" by some.[10] These consist of masses of cells that bear some resemblance to lymphocytes. In fact such tumors were considered to be sarcomas until some thirty years ago. They differ from lymphocytes in their greater irregularity, and in their frequently ovoid, rather than spherical, shape. The oat cells have finely stippled, rather densely staining nuclei, and scanty basophilic cytoplasm. Mitoses are frequent. Other tumors classified here as anaplastic consist of larger, more spindle-shaped cells with basophilic cytoplasm. From these, transitions can be traced to epidermoid tumors. Different sections of some tumors may show various degrees of anaplasia. Occasionally there is palisading at the margins of masses of the tumor cells. Large zones of necrosis may be visible histologically, but this tissue does not usually slough out *en masse*, and therefore grossly visible cavitation is distinctly unusual. In the reticulum stain the argyrophilic fibers are not generally found to penetrate into the masses of tumor cells.

Although many of these tumors may not be distinguishable in position and gross appearance from the epidermoid tumors, they do tend to predominate in the stem bronchi of each lung, and rarely occur beyond the lobar bronchi. True peripheral tumors of anaplastic type are distinctly uncommon. Typically the tissue is pale and translucent. Necrosis tends to be minimal and is rarely sufficient to result in gross cavitation. The nodes are massively involved even when the primary lesion is small, and this may occur at a time when

Fig. 16. Same patient. Film of May, 1954 at age of seventy-three. The mass has now grown much larger. At this time the patient complained only of mild cough productive of 2 to 3 tablespoons of non-sanguineous sputum.

Fig. 17. Abundantly keratinized squamous carcinoma with cavitation. The mass occupies the subsuperior and posterior basal segments.

Fig. 18. Anaplastic carcinoma. Right hilar mass continuous with a mediastinal mass that extends upwards toward the neck. Notching typical of carcinoma is visible on the lateral aspect. Collapse of the right lower lobe has not occurred, but there is a reticular appearance in the perivascular regions suggestive of lymphogenous spread. Compare with Figure 19.

Fig. 19. Anaplastic carcinoma involving the right stem bronchus, as seen from the posterior aspect. Many lymph nodes are involved, including the subcarinal, paratracheal, and the lymph node of the ductus arteriosus. The last mentioned is seen below the transected aorta. The shape of the tumor conforms to that observed in the roentgenogram. The characteristic notching is visible. The bronchus has become markedly narrowed, but is not completely obstructed. The generally well aerated substance of the right lower lobe is seen beneath the tumor, but there are streaks and minute nodules of gray tissue representing tumor within lymphatics.

the main bronchus has not as yet become obstructed. Involved lymph nodes and lymphatics extend in great collar-like masses proximally along the bronchial wall, beneath the carina and into the main mediastinal chains. Consequently they often present as hilar masses in which the shadows of the parenchymal mass and lymph nodes are continuous with each other and with that of the hilar structures (Fig. 18 and 19). Invasion of lymphatics also progresses peripherally about the bronchovascular rays and in the septa of the lung. Consequently the "sunburst" appearance typical of lymphogenous spread may be evident in the roentgenograms in late cases. Although often late in the disease, evidences of bronchial ob-

Fig. 20. Adenocarcinoma. Routine chest film in a thirty year old roentgen-ray technician, asymptomatic at this time. Film of March, 1952.

struction, when they occur, are usually massive.

Once symptoms exist and the diagnosis is made, the progress of the lesion is usually a rapid one and survival beyond the space of a year is uncommon. Although spread is predominantly via the lymphatic channels, hematogenous metastases are not uncommon. In fact one of the latter, for example a pathological fracture of a long bone, may be the first evidence of an anaplastic carcinoma.

Adenocarcinoma. Approximately 15 per cent of malignant bronchogenic tumors are adenocarcinomas. Although still preponderantly a disease of the male, adenocarcinoma is the most common type in women. Too little information is available at present to consider separately from ordinary adenocarcinomas the papillary, mucoepidermoid and other variants that have been described. Most adenocarcinomas, approximately 85 per cent, contain mucus demonstrable by special staining procedures. This feature is especially valuable when acini are inconspicuous.

Although most of the adenocarcinomas involve the segmental bronchi and their immediate branches, relatively more occur peripherally than is the case with other histopathological types.[13,17] These tumors ordinarily have a pearly gray translucent appearance, and the cut surface is finely flecked with black anthracotic pigment. Grossly visible foci of necrosis are generally small, and cavitation, although it may occur, is relatively uncommon. In particular, cavities in excess of 2 cm. are rarely encountered. A glairy cut surface, suggestive of a large content of mucus, is sometimes observed. This is only rarely en-

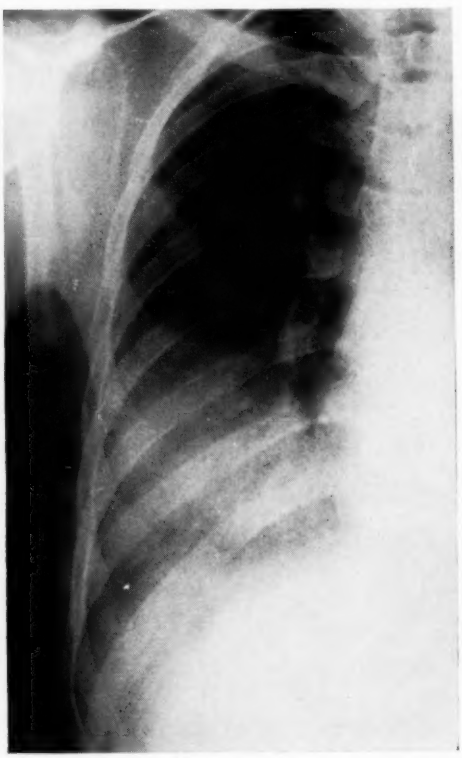

Fig. 21. Same patient as in Figure 20, ten months later. Film of January, 1953. The mass has increased enormously in bulk indicating a rate of growth much greater than that of the well differentiated squamous cell carcinoma in Figures 14–16. Compare with Figure 22.

countered in the gross. Despite the peripheral position of adenocarcinoma, the chest wall is not commonly involved before the tumor has otherwise manifested itself.

Data regarding the rate of growth of adenocarcinoma before it reaches a clinical level is difficult to obtain, but it would seem from serial films of "coin lesions" that had been unwisely "watched," that growth is rapid as contrasted with that of squamous cell tumors (Fig. 20–22). Hematogenous metastases tend to occur early and widely from otherwise silent pulmonary lesions. Their often peripheral position accounts for their insidious growth within the lung. Lymphatic pathways also are not neglected in metastasis.

Bronchiolar Carcinoma (*"Alveolar Cell Carcinoma," "Pulmonary Adenomatosis"*). Recent observations,[6,8,12,23,27] and especially the extensive summary of Storey, Knudtson and Lawrence,[26] have expanded knowledge of the natural history of peripheral acinar tumors variously called bronchiolar carcinoma, alveolar cell tumor, or pulmonary adenomatosis. These are epithelial neoplasms of various degrees of differentiation that seem so peripheral as not to be associated with any major bronchus. Although the evidence cannot be discussed here, the bulk of it indicates that these tumors arise in bronchioles rather than alveoli. These tumors are variously stated to comprise between 2 and 5 per cent of all pulmonary carcinomas. The incidence is approximately the same in the two sexes. As in the case of bronchogenic adenocarcinoma there is a very wide age distribution, but the median is in the middle fifties.

The epithelium varies from case to case and from place to place within the same tumor. In some, the cells are of the tall, columnar mucus producing type occurring in a single layer, with basally placed rounded or flattened nuclei. To this type the designation "pulmonary adenomatosis" has often been applied and the lesion resembles an infectious, probably viral, disease of sheep, jaagsiekte. Ciliated cells

Fig. 22. Adenocarcinoma. Same patient as in Figures 20 and 21. The peripherally situated bulky soft mass lies within the anterior basal segment of the right lower lobe. There are small foci of hemorrhage, but without massive cavitation. Lymph nodes containing metastatic tumor are shown within the hilus above and to the right.

are only rarely observed. Mitoses are extremely rare. These atypical cells may abut without transition upon normal ciliated low columnar lining cells of the bronchioles. Hutchison[12] has, however, described intermediate "premalignant" changes. In other instances, the epithelial cells are more variable, mucus is scanty or demonstrable only in an occasional cell, or it may be entirely absent. These cells may be quite bizarre, with irregular gigantic forms. The epithelium may be present in many layers, and may even come to fill the distal air spaces, while the stroma remains intact. All gradations between these two extremes exist even within a single tumor. Calcification, usually in the form of rounded lamellated psammoma bodies, occurs, surprisingly, in almost 50 per cent, but is usually not extensive and is not demonstrable roentgenographically. Despite these variations in epithelium there

Fig. 23. Bronchiolar carcinoma. Elongated bronchiole, met in almost longitudinal section, the lining epithelium of which has been almost entirely replaced by tumor cells. The wall is thickened by invading acini, and by the deposition of fibromuscular stroma containing small mononuclear cells. One branch terminates in a series of much smaller channels surrounded by an immense mass of stroma. The broad patency of the lumen proximally, the increase in the thickness and presumably in rigidity of the wall, and the apparently blind termination of some branches correlate well with the bronchographic findings in this disease as described by Zheutlin, Lasser and Rigler.[30]

appears to be no correlation with the clinical course; even the best differentiated tumors, such as have been designated "pulmonary adenomatosis," may metastasize. The lymphatics have been studied in an effort to obtain a clue as to the mechanism of spread within the lung, and tumor tissue has been found within them in over 50 per cent of these lesions. At times structures, for example, vessels, surrounded by these lymphatics appear to be compressed or even obliterated.

Recently Zheutlin, Lasser and Rigler[30] have described a bronchographic peculiarity of bronchiolar carcinoma. Within the zones of involvement, the small bronchi appear narrow, elongated, rectilinear, rigid, and filled rather than coated with lipiodol, without the usual "alveolar filling" beyond. They appear like a series of exclamation points. Histopathologically this may correlate with small bronchi and bronchioles whose lining epithelium has been replaced by tumor cells without obliteration of the lumen proximally, but whose walls have been thickened by desmoplastic reaction or lymphatic permeation by the neoplasm, or by lying surrounded by tumor filled alveoli (Fig. 23). Terminally the lumen of such a bronchiole may become criss-crossed by tumor embedded within abundant connective tissue and muscle that would interrupt the outward penetration of the lipiodol.

Grossly a nodular and a diffuse form have been described. Perhaps a better subdivision would be into discrete tumor-like and pneumonia-like forms. Actually closer inspection reveals that these two forms are not sharply different, and at times they are

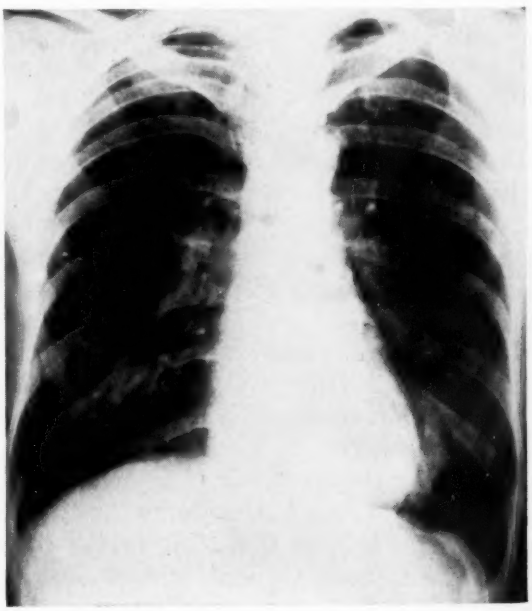

Fig. 24. Bronchiolar carcinoma. The patient had a history of chronic cough with expectoration of fourteen years' duration. In February, 1950, at the age of forty-four, a zone of infiltration in the left lower lobe and some accentuation of vascular markings in the right lung were noted in a chest film.

found in combination. The lesion may be encountered as a single peripheral focus, as mutliple foci, and as an apparently diffuse lesion involving a major portion of a lobe or an entire lobe.

In the more common nodular form, discrete nodules resembling metastatic tumor are scattered throughout the lung (Fig. 24–28), and indeed an extrapulmonary primary tumor elsewhere must be ruled out. Metastatic adenocarcinoma may closely resemble bronchiolar carcinoma as it involves both bronchioles and alveoli. In the nodular form of bronchiolar carcinoma there is usually no necrosis and this is in contrast with most metastatic tumors. A rich content of mucus may be discerned grossly, but is more apt to be obvious in the so-called diffuse form. The pleura is usually not invaded, but occasionally it may be puckered or actually invaded locally. Diffuse involvement by tumor, however, is very uncommon. Effusions and adhesions or both are observed in less than 20 per cent when the patient is first seen, but have a much higher incidence at death.

Fig. 25. Same patient as in Figure 24. Film of April 27, 1950. A small rounded shadow is now well defined on the right side.

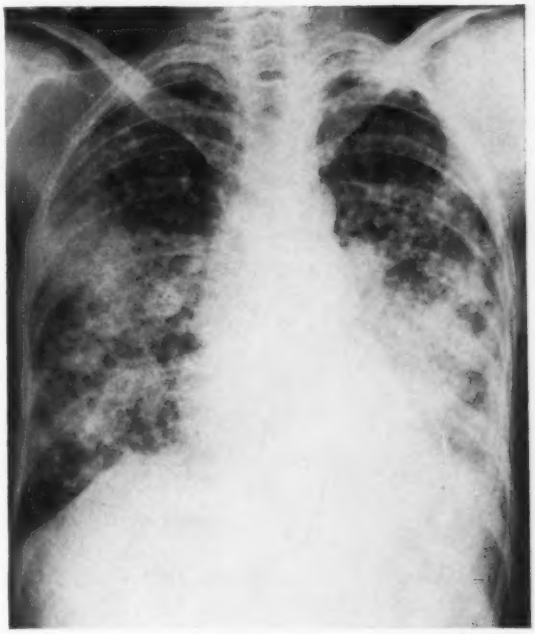

Fig. 26. Same patient as in Figures 24 and 25. Numerous minute nodules have appeared throughout both lungs, and seem confluent in the base of the left lower lobe (compare with Fig. 27), and in a wedge-shaped zone in the right upper lobe. Compare with Figure 28.

Less common than the focal nodular form, is the pneumonia-like or so-called diffuse form (Fig. 29–30). This may involve an entire lobe, or may exist in single or multiple foci. In the roentgenogram it often appears as a poorly circumscribed shadow or series of shadows suggesting pneumonitis. Involved tissue grossly has a resemblance to tuberculous pneumonia or to gray hepatization. Well differentiated mucus-producing tumors of the type referred to by some as pulmonary adenomatosis are especially apt to have this diffuse appearance. It is clear that the grossly diffuse lesion, as Musser, who described the first such case realized,[12] actually consists of large numbers of very small, closely placed, generally discrete lesions. There may be confluence in some foci. In part the gross and roentgenographic appearance of

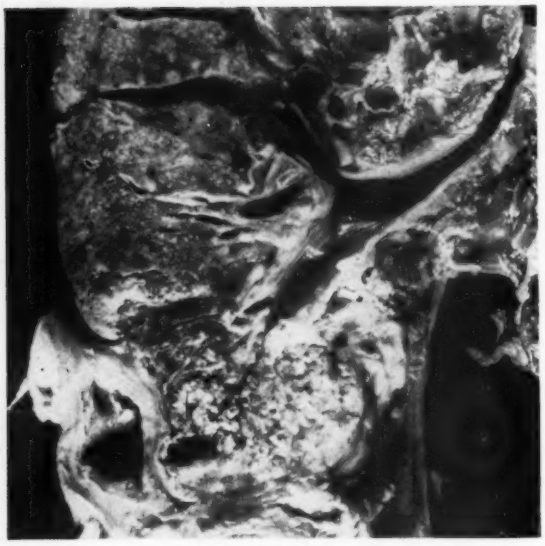

Fig. 27. Bronchiolar carcinoma. Posterior aspect of left lung shown in Figures 24–26. The mass is seen to consist of multiple nodules which have become confluent on the medial aspect of the lower lobe and just above the diagram. A small cavity has formed on the lateral aspect of this mass, and a small thick-walled pleural pocket is seen to the left of the intrapulmonary cavity. Numerous smaller nodules are scattered throughout the lung.

confluence and of vague definition from the surrounding parenchyma is given by mucus aspirated into the air passages even where these are uninvolved by tumor. This material may exude in large quantities when the tissue is incised, as a viscid glairy fluid and the cut surface may have a gelatinous appearance (Fig. 30). In this form, too, massive necrosis is not found, but necrosis may occur gradually with a more subtle crumbling of tissue and loss of substance, so that irregular cavities lined by well preserved tumor tissue result. These cannot usually be seen in the roentgenograms.

Statements regarding the natural history of these tumors depend upon the criteria employed in distinguishing peripheral bronchogenic carcinoma from bronchiolar carcinoma, and upon whether the experience of a large surgical center, or of an autopsy service is cited. In approximately 25 per cent of instances the tumor is first detected as a solitary nodule, but more than half of the patients have bilateral scattered lesions when first detected. Sequential roentgenographic studies on apparently isolated lesions have demonstrated that

Fig. 28. Same patient as in Figures 24–27. Many minute nodules are scattered above and below the obliterated fissure separating the right upper and middle lobes. The wedge-shaped zone of confluence corresponds to that shown in Figure 26. A small cavity is beginning to form.

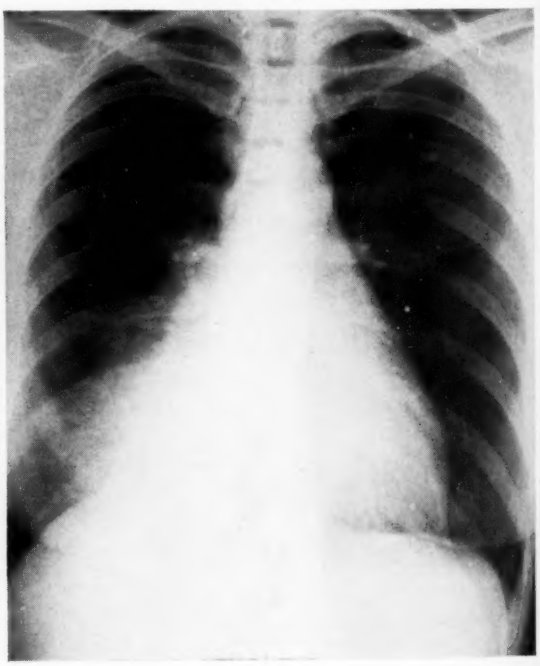

Fig. 29. Bronchiolar carcinoma, "diffuse" type. Infiltrative process involving the lower portion of the right lung.

some are remarkably stable and exhibit little or no evidence of growth over intervals of some years. Thus a young patient reported by Rosemond and his coworkers[21] was observed from 1944 to 1947 whereupon a resection was performed. There was no evidence of recurrence three years later. The isolated discrete lesion, of course, presents the best opportunity for effecting a surgical cure, and there have now been reported some half dozen patients who have survived in good health for five years following lobectomy or even more limited local resection.[21,25,26] From data currently available certain statements regarding prognosis may be made tentatively. It would appear that the pneumonia-like form has a less favorable prognosis than the grossly nodular form. If the disease is multiple, death usually occurs within two years, but if there is no evidence of recurrence within two years after resection, the patient is likely to remain well.[26]

Spread of the disease within the lung is not only by increase of the original lesion, but also very probably by aspiration and by lymphatics. The possibility of successfully transmitting experimental tumors by intratracheal inoculation has been demonstrated.[7] In more than 50 per cent of instances of bronchiolar carcinoma the lymphatics are obviously involved. In addition, the possibility that the tumor may arise in new independent foci within the lung has not been ruled out. Extrapulmonary lymphatic metastases, or hematogenous metastases, are observed terminally, again in approximately 50 per cent of patients. About half of the former are confined to intrathoracic lymph nodes. The others are similar to those observed in bronchogenic carcinoma. Most patients, however, die in respiratory failure from progressive replacement of pulmonary substance rather than from metastases. Cor pulmonale also may occur.

"Adenomas" and Cancer of the Lung. There has recently[5,19] been an outcry against the implications of the term "bronchial adenoma" as it has been generally applied. The objection has been particularly vehement in the case of the cylindromatous ("adenoid-cystic") tumor, as contrasted with the "carcinoid" type. The differences between these two lesions have also been emphasized, for example the more central position of the cylindromatous tumor, and especially its frequent position in the trachea or main carina, where the carcinoid tumor is very rarely encountered if at all. The greater invasiveness of the cylindromatous tumor, for example of such structures within the lungs as cartilage, is well known, as is the fact that both types are capable of metastasis, largely to hilar lymph nodes, but occasionally to distant organs such as the liver. According to a recent review,[5] the incidence

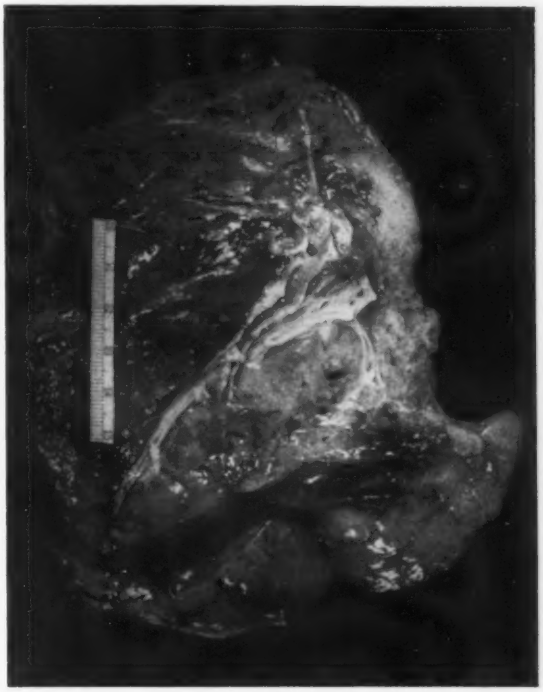

Fig. 30. Bronchiolar carcinoma, "diffuse" type. The resected right lower lobe from a distance appears completely consolidated. Upon closer inspection of the cut surface, especially on the mediastinal side near the base, immense numbers of very minute nodules are actually visible. These give the appearance of confluence at a distance. Much of the consolidation is from mucus aspirated into the parenchyma, to which it imparts a translucent glairy appearance and firm consistency.

of metastases in the cylindroid type is 32 per cent while in the carcinoid tumor it is 9.5 per cent. Prognosis for survival is much better with the latter. This, however, is largely the consequence of the technical difficulty of surgical treatment of tumors involving, or close to, the carina of the trachea. It has been argued that the cylindroid variety should therefore be considered carcinoma. It should be emphasized that the clinical differences between the two types are largely quantitative, and if the cylindroid tumor is to be considered malignant, so logically also should be the carcinoid.

These tumors, while not "benign," however, differ utterly in their development and course from bronchogenic carcinoma. The history of symptoms of pulmonary disease is very much longer with bronchial adenoma, and may extend over decades, and the curability with surgery is much higher. Moreover the prognosis is little altered by the presence of metastases in hilar lymph nodes, and when tumor is of necessity left behind, life may go on for many years. Palliative resection to clear the airways may be repeatedly performed. Death from widespread metastases occurs in less than 3 per cent.

If these differences from bronchogenic carcinoma are clearly recognized, the semantic problem assumes little importance. It is clear that in the scheme of nature there are some neoplasms that differ from adenoma, yet metastasize and invade less vigorously than do most to which the appellation carcinoma is applied. To this group belong the tumors that had best be called bronchial carcinoid or cylindromatous tumors, without reference to "adenoma" or "carcinoma."

SUMMARY

Some aspects of the relationship between histopathologic structure and natural history of primary carcinoma of the lung, and especially of pathology in relation to the roentgen shadow, are discussed. Thus a hilar tumor with evidence of massive lymph node involvement is likely to be an anaplastic carcinoma; a peripheral tumor in a young woman, adenocarcinoma; and a peripheral tumor, especially when demonstrably cavitated, that invades the chest wall in a man over sixty, a well differentiated squamous cell carcinoma. Considerable overlap exists, however, in these and other features.

Evidence, unfortunately not conclusive, exists that tends to support: (1) the bronchiolar origin of tumors known otherwise as pulmonary adenomatosis, or alveolar cell carcinoma; (2) that, while some of these tumors certainly arise in a single focus and are therefore potentially curable by surgery, others may originate in multiple foci; (3) that there is little fundamental difference between so-called nodular and diffuse or pneumonia-like forms; (4) that aspirative spread is possible; (5) that even the best differentiated of these tumors appear to pursue a course no different from from others in the group.

The differences between lesions generally called bronchial adenoma and bronchogenic carcinoma are stressed, and especially that even the cylindromatous (adenoid-cystic) type of the former rarely causes death by metastasis. It is suggested that they simply be called bronchial carcinoid and cylindroma respectively, thereby avoiding a semantic quarrel, and admitting the existence of tumors that occupy an intermediate position between adenoma and carcinoma.*

Yale University School of Medicine
New Haven, Connecticut

REFERENCES

1. CAHAN, W. G., WATSON, W. L., and POOL, J. L. Radical pneumonectomy. *J. Thoracic Surg.*, 1951, *22*, 449–473.
2. CARLISLE, J. C., MCDONALD, J. R., and HARRINGTON, S. W. Bronchogenic squamous-cell carcinoma. *J. Thoracic Surg.*, 1951, *22*, 74–82.
3. CUDKOWITZ, L., and ARMSTRONG, J. B. Blood supply of malignant pulmonary neoplasms. *Thorax*, 1953, *8*, 152–156.
4. DOTTER, C. T., STEINBERG, I., and HOLMAN,

* For discussion see page 436.

C. W. Lung cancer operability; angiocardiographic study of fifty-three consecutive proved cases of lung cancer. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 222–238.

5. Enterline, H. T., and Schoenberg, H. W. Carcinoma (cylindromatous type) of trachea and bronchi and bronchial adenoma. *Cancer*, 1954, *7*, 663–670.
6. Fisher, J. H., and Holley, W. J. Primary alveolar cell carcinoma of lung. *Arch. Path.*, 1953, *55*, 162–170.
7. Furth, J. Experiments on the spread of neoplastic cells through respiratory passages. *Am. J. Path.*, 1946, *22*, 1101–1107.
8. Good, C. A., McDonald, J. R., Clagett, O. T., and Griffith, E. R. Alveolar cell tumors of lung. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 1–19.
9. Griess, D. F., McDonald, J. R., and Clagett, O T. Proximal extension of carcinoma of lung in bronchial wall. *J. Thoracic Surg.*, 1945, *14*, 362–368.
10. Halpert, B. and Pearson, B. Cellular structure of carcinoma of lung. *Am. J. Cancer*, 1940, *40*, 213–218.
11. Herbut, P. A., and Watson, J. S. Tumor of thoracic inlet producing Pancoast syndrome; report of seventeen cases and review of literature. *Arch. Path.*, 1946, *42*, 88–103.
12. Hutchison, H. E. Pulmonary adenomatosis and alveolar-cell carcinoma; review. *Cancer*, 1952, *5*, 884–907.
13. Liebow, A. A. Tumors of the Lower Respiratory Tract. Fascicle 17. Atlas of Tumor Pathology. Armed Forces Institute of Pathology, Washington, 1952.
14. Lindskog, G. E., and Liebow, A. A. Thoracic Surgery and Related Pathology. Appleton-Century-Crofts, New York, 1953.
15. McBurney, R. P., McDonald, J. R., and Clagett, O. T. Bronchogenic small-cell carcinoma. *J. Thoracic Surg.*, 1951, *22*, 63–73.
16. Pancoast, H. K. Superior pulmonary sulcus tumor; tumor characterized by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. *J.A.M.A.*, 1932, *99*, 1391–1396.
17. Patton, M. M., McDonald, J. R., and Moersch, H. J. Bronchogenic adenocarcinoma. *J. Thoracic Surg.*, 1951, *22*, 83–87.
18. Patton, M. M., McDonald, J. R., and Moersch, H. J. Bronchogenic large-cell carcinoma. *J. Thoracic Surg.*, 1951, *22*, 88–93.
19. Reid, J. D. Adenoid cystic carcinoma (cylindroma) of bronchial tree. *Cancer*, 1952, *5*, 685–694.
20. Rigler, L. G., O'Loughlin, B. J., and Tucker, R. C. Duration of carcinoma of lung. *Dis. Chest*, 1953, *23*, 50–71.
21. Rosemond, G. P., Boucot, K. R., and Aegerter, E. E. Solitary pulmonary adenoma (focal pulmonary adenomatosis): three-year follow-up after resection. *J. Thoracic Surg.*, 1951, *22*, 99–103.
22. Rouvière, H. Anatomy of the Human Lymphatic System. Translated by M. J. Tobias. Edwards, Ann Arbor, 1938.
23. Slesser, B. V., Britt, R. G. and Freer, J. L. Assessing inoperability of bronchial carcinoma by angiocardiography. *Thorax*, 1954, *9*, 91–105.
24. Smetana, H. F., Iverson, L., and Swan, L. L. Bronchogenic carcinoma; analysis of 100 autopsy cases. *Mil. Surgeon*, 1952, *111*, 335–351.
25. Smith, R. R., Knudtson, K. P., and Watson, W. L. Terminal bronchiolar or "alveolar cell" cancer of lung. *Cancer*, 1949, *2*, 972–990.
26. Storey, C. F., Knudtson, K. P., and Lawrence, B. J. Bronchiolar ("alveolar cell") carcinoma of the lung. *J. Thoracic Surg.*, 1953, *26*, 331–406.
27. Watson, W. L., and Smith, R. R. Terminal bronchiolar or "alveolar cell" cancer of lung; report of 33 cases. *J.A.M.A.*, 1951, *147*, 7–13.
28. Weinberg, J. A. Identification of regional lymph nodes in treatment of bronchiogenic carcinoma. *J. Thoracic Surg.*, 1951, *22*, 517–526.
29. Wright, R. D. Blood supply of abnormal tissues in lungs. *J. Path. & Bact.*, 1938, *47*, 489–499.
30. Zheutlin, N., Lasser, E. C., and Rigler, L. G. Bronchographic abnormalities in alveolar cell carcinoma of lung. *Dis. Chest*, 1954, *25* 542–549.

THE DETECTION OF CARCINOMA OF THE LUNG BY SCREENING PROCEDURES, PARTICULARLY PHOTOFLUOROGRAPHY*

By L. H. GARLAND, M.B.
SAN FRANCISCO, CALIFORNIA

SCREENING procedures may be defined as preliminary diagnostic examinations applicable primarily to asymptomatic persons. For purposes of detecting lung cancer, they might conceivably consist of (a) biochemical methods, (b) cytologic methods, or (c) roentgenologic methods. There is no acceptable biochemical method to date, although considerable work with a "seroflocculation reaction" is currently in progress.[13] Mass surveys for cancer in other sites, using cytologic methods, have not proved practical.[25] We are therefore left with roentgenologic methods as the best available tool at the present time.

The most commonly utilized screening procedure is one employing single, anterior exposures made in full inspiration on 70 mm. film. However, film of various sizes ranging from 35 mm. roll film to individual 14 by 17 inch film has been used; paper media and roentgenoscopic methods have also employed. Multiple exposures have been recommended, but not yet carried out in any large survey. Extensive investigations by the author and his associates have confirmed the fact (first shown by Birkelo, Chamberlain, *et al.*)[2] that the *size* of film employed for the detection of abnormal pulmonary shadows in mass surveys is of less importance than the "personal equation" of the physician interpreting the films.[14,15] These observations have also been verified by Cochrane and his associates in England,[8] and by Groth-Petersen and his associates in Denmark.[17] In other words, for mass survey purposes any size of properly exposed film is equally reliable for the detection of abnormal pulmonary shadows. The three most important factors are correct technique, careful interpretation and the use of dual readings.[38] Readers should be trained, experienced and preferably pre-tested. If one measures error by the number of "positives" missed, we have shown that in mass surveys for the detection of pulmonary densities of inflammatory origin:

Experienced readers miss about 27 per cent of cases;
Less experienced readers miss 43 per cent of cases;
Inexperienced readers miss over 50 per cent of cases.[15]

This degree of error doubtless holds true also for the detection of shadows of neoplastic origin. Churchill[7] analyzed the results of the San Diego Mass Chest X-ray Survey and found that at least 6 of 26 cases of bronchogenic carcinoma were overlooked (23 per cent). Boucot and Sokoloff[4] found in the Philadelphia Public Health Survey that 11 of 77 cases of lung tumor were completely missed (14 per cent), and 36 of 77 were either missed or labeled "tuberculosis only" (47 per cent). These two groups of "misses" were discovered on retrospective review of the photofluorograms.

The types of pulmonary lesion commonly overlooked in minifilm surveys have been shown to include the following:

Densities less than 2 cm. in diameter;
Densities in the peripheral portions of the lungs;
Densities lying behind bony or soft tissue structures, especially close to the mediastinum and at the bases;

* From the Stanford University Department of Radiology, San Francisco Hospital, San Francisco, California. Presented in a Symposium on Carcinoma of the Lung at the Fifty-fifth Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

Partly obstructive lesions with local emphysema (visible only in expiratory films);

Densities masked by associated pneumonitis, tuberculosis, or cavitation.[15,17]

In surveys of small groups, stereoscopic anterior projections have been utilized and are naturally preferable to single projections for several obvious reasons. In quite small groups some workers have also used frontal views in inspiration and expiration plus lateral views in inspiration. All of these provide additional opportunity for detecting lesions obscured by osseous and other structures, and for detecting small areas of emphysema, subsegmental atelectasis, and so forth. However, for true mass screening purposes, one must still rely principally on single, frontal projections, utilizing minifilms.

It has been shown by Pendergrass and his associates that ultrahigh voltage technique (about 2,000 kv.) permits the recording of small densities obscured by osseous and mediastinal shadows with conventional voltages (up to 125 kv.). However, no screening programs have yet been reported utilizing megavoltage technique.

The follow-up roentgen examination on the "tumor suspect" cases detected in the survey should be adequate in scope and accompanied when indicated by roentgenoscopic examination. It is advisable to make stereoscopic anterior films in inspiration, plain anterior in expiration, and a lateral film. Laminagrams should be used when necessary, especially to aid in locating and identifying questionable calcific deposits in a solitary lesion. Lordotic, oblique and other studies should be employed in appropriate cases. That this "follow-up" examination should be prompt, as well as thorough, goes without saying, even though this desideratum is often difficult of attainment.

TYPES OF BRONCHOGENIC CARCINOMAS RECORDABLE BY SCREENING PROCEDURES

The gross morphologic types of bronchogenic carcinoma recordable on photofluorograms depend on the size and location of the tumor, and the presence or absence of associated pulmonary collapse, infection, adenopathy or pleural effusion.

Direct evidence or the *tumor* itself is recordable when it presents as a visible mass partly or completely surrounded by air-bearing parenchyma. It may show central necrosis with cavitation.

Indirect evidence of the tumor is manifest by disturbed bronchial function (with secondary infection or *pneumonitis*), partial bronchial blockage (with obstructive *emphysema*), and complete bronchial blockage (with *atelectasis*). *Adenopathy* or *pleural effusion* may be the first manifestation.

It has been found in some studies that about 50 per cent of primary lesions are situated in portions of the lungs readily visible on frontal projections. However, Boucot and Sokoloff in a recent study found that in only 25 per cent of their 77 cases were the tumors so located. They noted that even then small peripheral lesions were readily overlooked when they lay behind bony structures.

Therkelsen[36] noted that in 64 per cent of a series of 329 cases of bronchogenic carcinoma the lesions manifested themselves in the initial roentgenogram by atelectasis; De Bakey[9] found that in about 62 per cent of cases the tumors occurred at or near the hilus. On the other hand, Bondi and Leites[3] found that only 3 of 21 survey-detected cases were central; 18 were peripheral, 4 of them "very small, not exceeding 1.5 cm. in diameter."

Both directly and indirectly recorded tumors may be complicated or masked by associated intrathoracic disease, such as active pulmonary tuberculosis. Approximately 8 per cent of four different series of cases of bronchogenic carcinomas were thus complicated in groups referred to by Boucot and Sokoloff.[4] We have seen several patients with combined active tuberculosis and lung cancer at the San Francisco Hospital.

Under ideal roentgenographic conditions, very small opacities of sufficient

TABLE I

FINAL CLASSIFICATION OF "TUMOR SUSPECT" CASES DETECTED IN MASS SURVEYS

Cases Suspect on Standard Film	Primary Lung Tumor (malignant)	Metastatic Lung Tumor (from extra-thoracic cancer), and Lympho-blastoma	Granuloma and Other Inflamm. Processes (tbc., sarcoid, pneumonia, fibrosis)	Miscellaneous Cancer Excluded, (benign tumor of lung or rib, cyst, cardiovascular, lesion, pleural or diaphragm abnormality, goiter, etc.)	Undetermined (chiefly awaiting follow-up)
	per cent	per cent	per cent	per cent	per cent
Bondi and Leites, New York 184 cases	15	13	14	26	32
Scamman, Boston 398 cases	12	5	→ 65 ←		18
Guiss, Los Angeles 3,500 cases	4	3	?*	20	29

* Guiss uses the term "chest neoplasm ruled out" for 1,537 cases or 44 per cent of the total 3,500 tumor suspects. This 44 per cent plus the 20 per cent miscellaneous group in his series corresponds well with the 65 per cent of similar cases in Scamman's series.

density or contrast can be recorded on the roentgenogram. However, for practical purposes, primary lung tumors smaller than 1 cm. are difficult to detect in the average photofluorogram. After consulting experienced pathologists we conclude that an average bronchogenic carcinoma 1 cm. in diameter contains about 33,000,000 cancer cells.* Therefore, when tumors are seen directly on the photofluorogram, they are apt to be at least of the above size; when recorded indirectly (by virtue of the secondary changes) they may be smaller or larger than that size.

Finally, and most important, extensive mediastinal adenopathy may be present but not recordable even on the most carefully exposed roentgenograms. Such adenopathy may be present very early in the case of anaplastic tumors and, conversely, may be quite late in the case of well-differentiated tumors. In one recent year on the Stanford X-ray Service at the San Francisco Hospital, we had 3 patients come to autopsy with metastatic bronchogenic carcinoma in the brain or abdominal viscera while the primary lesion was not demonstrable on standard pre-mortem roentgenograms. In all 3 cases the primary lesion was situated close to the mediastinum, being obscured by cardiovascular or osseous structures.

The proportion of "tumor suspects" detected by roentgenologic surveys who actually prove to have primary carcinoma of the lung varies in different series. While Overholt and Atwell[31] stated that about 40 per cent of silent lesions suspected as tumor out to be cancer, Scamman[33] in the Boston survey found that only 12 per cent

* The diameter of the "average" cancer cell in a majority of bronchogenic carcinomas is about 25 μ. The range is from about 10 to 40 μ. If the primary tumor is a peripherally located sphere or coin lesion, composed of lung cancer cells of average diameter, a lesion *1 cm.* in diameter will contain about *33 million cancer cells;* a tumor 2 cm. in diameter will contain about 260 million cancer cells. A "small" cancer is therefore rarely an "early" cancer.

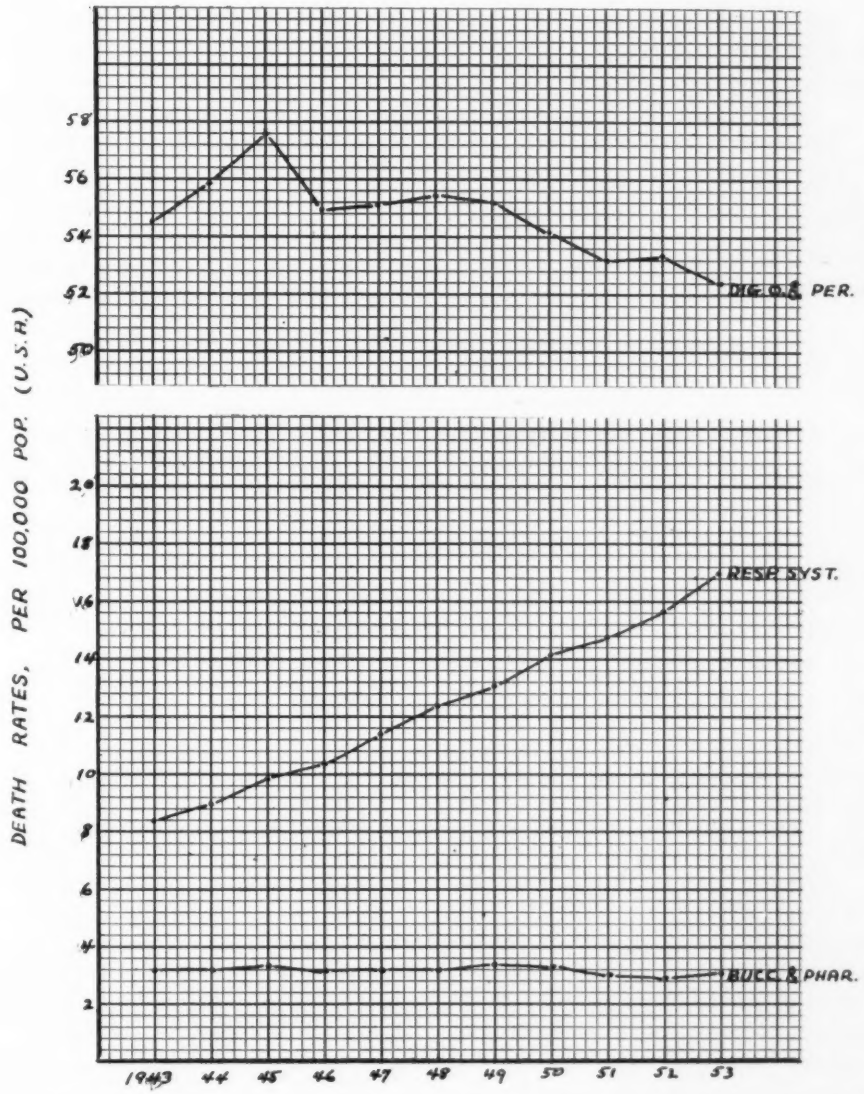

FIG. 1. Reported Death Rates per 100,000 Population for Certain Malignant Neoplasms (United States).

The upper curve shows the reported death rates from malignant neoplasms of the digestive organs and peritoneum for the years 1943 to 1953, inclusive. The middle curve shows the reported death rates from malignant neoplasms of the respiratory system; as may be noted from the text, approximately 75 per cent of these deaths in recent years have been due to bronchogenic carcinoma. The lowest curve shows the reported death rates from malignant neoplasms of the buccal cavity and pharynx.

Part of the increase for respiratory system cancers is probably illusory, due to such factors as *better diagnoses, more frequent reporting* of lung cancers as primary rather than secondary (both during life and at necropsy), and increasing *aging* of the population.

Source: U. S. Vital Statistics Report. Vol. II, June 18, 1954. U. S. Department of Health, Education, and Welfare, Washington, D. C.

proved to be such; Bondi and Leites[3] in New York 15 per cent, and Guiss[18] in Los Angeles 4 per cent (Table I).

If "solitary pulmonary lesions" alone are considered, the proportion that proved to be cancer was somewhat higher, averaging about 24 per cent in a series of 10 reported groups collected by May *et al.* (Table II).

In non-survey material, Hood *et al.*[22] found that 16 per cent of a series of 156

TABLE II

PATHOLOGIC FINDINGS IN TEN SERIES OF CASES OF SOLITARY PULMONARY LESIONS

(Total Cases 336)

	Cases	Per Cent
Bronchogenic cancer	82	(24)
Metastatic malignant lesion (incl. lymphomas)	26	(8)
Benign tumor	4	(1)
Granuloma	140	(42)
Other process	84	(25)

lesions in patients with solitary circumcribed shadows seen at the Mayo Clinic proved to be bronchogenic carcinoma (Table III).

It is probably a reasonable estimate that about 25 per cent of clinically silent solitary lesions in *men over forty-five* are due to cancer.

INCIDENCE OF BRONCHOGENIC CARCINOMA

The incidence and prevalence of bronchogenic carcinoma must be determined partly on the basis of death certificates, the limited reliability of which is known to all students of the problem. However, it has been contended that the number of erroneous over-diagnoses of bronchogenic carcinoma on death certificates is balanced by the number of persons actually dying from that disease, but erroneously recorded as dead of other causes. Official vital statistics list bronchogenic carcinoma deaths along with other malignant neoplasms of the respiratory system.[37] The official estimated United States death rate from this group of diseases in 1953 was 17 per 100,000. According to the Metropolitan Life Insurance Company, death rates from respiratory cancer show wide variation over the United States.[34] In general, they are highest in the Northeast and lowest in the South, the maximum rates among white males being approximately *six* times the minimum figures. These geographic variations reflect differences in age, sex, diagnostic resources and other factors.

Horn[23] (Assistant Director of Statistical Research for the American Cancer Society) states that there were approximately 22,000 deaths from primary carcinoma of the lung in the United States population of some 160,000,000 persons in 1953. This is equivalent to a death rate of *14* per 100,000. Guiss[20] has made a painstaking survey of the Los Angeles area during the last five years and finds that the death rate in that part of the country is *13* per 100,000.

Official United States mortality data for *lung cancer* are not synonymous with *respiratory tract cancer*, although the rates for the latter are often quoted as being essentially those for bronchogenic carcinoma. The most recent available final statistics are for the year 1951. These read as follows:

"Malignant neoplasms of respiratory system (Items 160–165 of the International Statistical Classification of Causes of Death):

160. Malignant neoplasm of nose, nasal cavities, middle ear and accessory sinuses	607	(0.4)
161. Malignant neoplasm of larynx	1,835	(1.2)
162. Malignant neoplasm of trachea, and of bronchus and lung, *specified* as primary	8,438	(5.5)
163. Malignant neoplasm of lung and bronchus, *unspecified* as to whether primary or secondary	11,287	(7.4)
164. Malignant neoplasm of mediastinum	346	(0.2)
165. Malignant neoplasm of thoracic organs (secondary)	325	(0.2)
Total	22,828	(14.0)

The figures in parentheses after the number of deaths are the rates per 100,000 population based on mid-year estimates."

TABLE III

PATHOLOGICAL CLASSIFICATION OF SOLITARY CIRCUMSCRIBED SHADOWS SEEN IN ROENTGENOGRAMS (HOOD ET AL)

Material: 1,355 patients received pulmonary resection (1940–1951). Cases with solitary circumscribed shadows: 156

	Cases	Per Cent
Bronchogenic carcinoma	25	(16)
Metastatic malignant lesions	17	(11)
Benign tumors	42	(27)
Granuloma	65	(42)
Other process incl. bronchial cysts	7	(4)

Table IV

	Per 100,000 pop. Both sexes	Per 100,000 Males	Per 100,000 Females
All ages	*12*	(19	4)
Ages 45–75	43	(71	15)

If we assume that all cases in category 162 are bronchogenic (8,438 cases), and that 80 per cent of those in category 163 are such (9,029 cases), it appears that out of the entire group of "respiratory system neoplasms" not more than 17,467 are probably primary lung cancers. The total figure for "respiratory system neoplasm" deaths should therefore be reduced by a factor of about one-fourth to obtain the "bronchogenic neoplasm" deaths.

Many charts published by the American Cancer Society show data for "respiratory system cancer except larynx." It is evident that even these data are incorrect for bronchogenic cancer, and should be reduced by a factor of about one-sixth.

The *mortality rate* for primary cancer of the lung in the United States per 100,000 population, age-adjusted, was recorded by Dorn as *12* for the year 1950. The rates for different ages and sexes for that year may be summarized as follows (Table IV).

The *incidence rate* (*i.e.*, the number of newly diagnosed cases of primary lung cancer per 100,000 living persons) was calculated by Dorn for that year as follows (Table V).

These incidence rates correspond reasonably well with those found in the U. S. Public Health Survey of ten cities for the year 1948, which were:

Atlanta	8.9
Dallas	16.3
Birmingham	13.6
Denver	14.8
New Orleans	20.8
Detroit	19.0
Pittsburgh	15.7
Chicago	15.9
Philadelphia	18.3
San Francisco	20.8

Table V

All ages	*18*	(30	6)
Ages 45–75	64	(108	20)

These rates range from 9 to 21, the average being *16*. If the same ratio of increase continued between 1948 and 1954 as existed in the prior decade, the average *incidence rate* for the above ten cities in 1954 should be about 24 per 100,000.

The *prevalence rate* (*i.e.*, the number of persons living at any one time with primary lung cancer, per 100,000 living persons) is reported at widely differing levels in different cities. Steiner[35] estimated the 1950 rates for San Francisco and New Orleans as about 25 for all ages. Since the average survival time in bronchogenic carcinoma is only twelve months after the onset of symptoms (and nine months after diagnosis), it is probable that the prevalence rate in general is pretty close to the incidence rate.[35]

The factor of age is strikingly shown in the figures given by Doll[11] from the British Ministry of Health. A study of mass roentgenography of some 1,500,000 persons per annum revealed in English males the following prevalence rates for reported bronchogenic carcinoma:

Ages 45–60	70 per 100,000 males
Ages over 60	200 per 100,000 males

Most of the preceding figures refer to more or less unselected, general population groups. If selected groups are considered, the prevalence rate will show corresponding adjustment.

Results of Screening Procedures

The results of screening procedures may be measured in terms of "tumor suspects detected," or "tumors detected," or "tumors detected and successfully treated." As far as cancer control is concerned, the latter is obviously the important yardstick.

The results of several "general population" and "selected group" surveys have been published. In the general population surveys, the average yield has been about

TABLE VI

BRONCHOGENIC CARCINOMA FOUND IN CHEST SURVEYS: GENERAL POPULATION SURVEYS

Reference	City	Persons Surveyed	Tumor Suspects, on Survey Film (and rate per 100,000)	Tumor Suspects, on Standard Film (and rate)	Broncho. Ca. Verified (and rate)	Broncho. Ca. Resected Cases	Survived 2–3 Yr. Cases
Overholt[30]	Several in U. S.	1,780,178 (9 city wide surveys)	—	1,382 (77)	— (10)	—	—
McNulty[26]	Boston	536,012 (gen. pop.)	—	398 (74)	39 (7.4)	22	5
Churchill[7]	San Diego	245,061 (gen. pop.)	—	—	20 (8)	4	2
Guiss[19]	Los Angeles	1,867,201 (gen. pop.)	64,745 (3,467)	3,500 (188)	222 (11.8)	84	24

10 verified bronchogenic carcinomas per 100,000 persons examined. Considering the fact that many of these persons were in the younger age groups or of the female sex, that not a few of the "tumor suspect" cases failed to return for verification of diagnosis or for treatment, and that not more than about 50 per cent of primary lung cancers are detectable on routine minifilms,* this

* The prevalence rate for bronchogenic carcinoma in the general population of Los Angeles, Boston and New York at the times of the large x-ray surveys performed in those cities was between 18 and 20 per 100,000. The detection rate finally reported was about 10 per 100,000 persons. Therefore about one-half the tumors were not detected, presumably due to many factors, including misses due to small size of tumor.

TABLE VII

BRONCHOGENIC CARCINOMA FOUND IN CHEST SURVEYS: SELECTED GROUP SURVEYS

Reference	City	Persons Surveyed	Tumor Suspects, on Survey Film (and rate per 100,000)	Suspects, on Standard Film (and rate)	Broncho. Ca. Verified (and rate)	Broncho. Ca. Resected, Cases	Survived 3 Yr.
Bondi and Leites[3]	New York City	228,375 (certain adult groups)	184 (81)	—	21 (9)	12 (9)	
May *et al.*[27]	Oakland	40,000 (V.A.H. admissions)	60 (150)	—	8 (20)	—	?
Boucot and Sokoloff[4]	Philadelphia	142,156 (chest clinic clients)	—	—	48 (30)	16	?

detection rate of 10 is reasonably proportionate to the estimated prevalence rate of 18 per 100,000 for the years in question (Table VI).

In the "selected group" surveys the yield has been naturally higher (about 20 per 100,000) (Table VII).

The above figures apply to verified bronchogenic carcinomas. If one considers the "tumor suspect" group as a whole, the yield in general population surveys in the United States for the decade ending 1950 has ranged as follows (Table VIII).

The classification of a group of "tumor suspects" one year after completion of a mass survey was shown by Guiss in his 1952 paper[18] as follows:

Total tumor suspects	3,500
Tumor "excluded on recheck"	1,537
Tumor (primary) "confirmed on recheck"	240
Metastatic lung tumor	89
Enlarged thyroid	417
Tumor, clinically benign, not treated	206
Treatment refused	74
Cases lost	108
Cases still under investigation	829

In the tumors which were confirmed (240 in number), there were malignant tumors, 177; and benign tumors, 63. The malignant tumors were made up of 144 carcinomas, 28 sarcomas or lymphomas, and 5 unspecified tumors. The benign tumors were mostly neurofibromas and benign lung tumors "unspecified."

In other words, as a result of this ten-month survey in Los Angeles involving almost 2 million people, 144 primary cancers of the lung were confirmed, or 7.6 cases per 100,000 persons.*

TABLE VIII

Region	Persons Examined	"Tumor Suspect" Rate Per 100,000 Persons
Milwaukee, Wis.	176,469	20
Tacoma, Wash.	72,703	40
Savannah, Ga.	67,961	60
Spokane, Wash.	106,526	60
Gaston, N. C.	84,599	70
Seattle, Wash.	368,129	70
Salt Lake City, Utah	162,351	70
Los Angeles, Calif.	1,867,201	188
Washington, D. C.	439,927	80
Minneapolis, Minn.	301,513	130
Boston, Mass.	536,012	74

TABLE IX

RESECTABILITY RATES

Survey Detected Tumors (mostly asymptomatic)

	Cases	Resectable
Guiss (Los Angeles)	222	39%
McNulty (Boston)	39	56%
Overholt (Boston)	67	60%
Bondi (New York)	21	50%
Boucot (Philadelphia)	48	40%
Denk (Vienna)	41	22%

Tumors Detected in Regular Practice (mostly symptomatic)

	Cases	Resectable
Jones (Los Angeles)	196	20%
DeBakey (New Orleans)	948	27%
Overholt (Boston)	721	28%
Moore (New York)	370	23%

RESULTS IN TERMS OF RESECTABLE TUMORS

The results of the screening procedures in the surveys to date show that the radical or curative resectability rate of the tumors detected ranged from about 22 per cent to 60 per cent. This is better than that of non-survey detected cases, although the number of patients in three of the groups is rather small for reliable conclusions. It is of interest to note that Denk[10] (in an experience of some 1,145 patients with bronchogenic carcinoma) analyzed 41 silent cancers of the lung and found that while 17 were potentially resectable, only 9 could actually be resected (22 per cent) (Table IX).

* Since publication of that paper, an additional group of primary carcinomas has been identified as of May, 1954, leading to a total yield of 222 cases to date. This higher figure has been used in all of the tables in this paper.

TABLE X

"CURABILITY" RATES OF SURVEY-DETECTED LUNG CANCER

	Cases Detected	Resected	Survival 3 Years	Per Cent of Cases Detected Who Survived 3 Years
Guiss (Los Angeles)	222	84	24	11
McNulty (Boston)	39	22	5	13
Overholt (Boston)	30	26	9	30
Churchill (San Diego)	20	4	2*	10
Total	311	136	40	12.8

* 2 years.

RESULTS IN TERMS OF POTENTIAL CURES

The *survival rates* in the resected cases of survey origin have been reported only for short periods of time. Guiss[20] reports that 24 of their 84 resected cases were alive at three years. McNulty[26] reports that 5 of their 22 cases are living at three years. Churchill[7] noted 2 of 4 resected cases in the San Diego area were alive at two years (out of 20 with verified primary tumors). *Therefore, of 281 with verified bronchogenic cancers detected in these three general population surveys, 110 were resected and some 31 patients survived approximately three years to date, a survival rate of 11 per cent of the cancers found by survey.** This is not significantly different from that obtained in regular practice (the two year absolute survival rate in DeBakey's series of 948 cases being 10 per cent and the five year rate 6.5 per cent) (Table X).

It may be argued that regularly repeated surveys will yield more worthwhile results. This has not been shown to date. Hemlin[21] recently reviewed the results of periodic roentgen examinations (annual or more often) of 10,000 employees, mostly male and all asymptomatic. The average annual yield was surprisingly high, 5 lung cancers; the cumulative yield in five years was 26 cases of lung cancer. The following are the results of treatment in these 26 cases:

Dead	18 (80%)
Disabled, sick or retired	6
Terminated work	1
Working	1 (4%)

COMPLICATIONS

The mortality rates in the resected groups varied considerably, both in the "cancer confirmed" and the "other disease" cases. For example, in the Los Angeles survey there were 70 "curative resections" in the carcinoma group with an operative mortality of 10 per cent, and 117 resections in the "other disease" group with a surgical mortality of 3.4 per cent. The latter 4 cases with non-cancerous lesions therefore lost their lives as an indirect result of the survey. There were 24 potential cures in the cancer cases inspected, so that the net yield of "cancer" lives saved by this survey was about 20 persons. McNulty did not mention operative mortality. Bondi and Leites reported one of 20 per cent; DeBakey reported a recent operative mortality rate of 18 per cent. It is recognized that in leading institutions, with expert surgery, the mortality rate of resection is now decreasing to less than 10 per cent.

A complication of surveys which is difficult to combat is the false sense of security assumed by many persons when the minifilm is reported as negative. Some such persons develop symptoms of a

* Overholt recently reported[22] that 9 of 30 survey-detected primary lung cancers survived three years (30 per cent). The resectability rate was 50 per cent in this group.

bronchogenic lesion but delay going to their physician because "their x-ray was all right." That x-ray may have been made a year ago. If they now harbor a tumor their delay may lead to further growth, with resultant inoperability. We have seen at least 2 such cases.

DISCUSSION

Several writers[6,30,31] have recommended that chest screening for cancer should be aimed primarily at select groups; for example males over forty-five. In actual practice it is questionable if such can be arranged. Young people predominate in most large employed groups, and participate in community surveys more readily than the elderly. Further, it is not possible to exclude males of other ages, nor females, who request examination.

However, if surveys could be restricted, it is probable that the following approximate ratios would apply currently to males over forty-five per 100,000 surveyed (Table XI).

These figures correspond closely to those calculated by Horn for the United States in 1953, namely 1,250 "tumor suspects" per 100,000 males over forty-five by survey, yielding about 50 primary bronchogenic carcinomas. The prevalence rate in males over forty-five is about 108. Survey examinations should lead to detection of about one-half of these, or 50–54 cases per 100,000 asymptomatic men in that age group.

In other words, about 1 minifilm per 100 will be recorded "tumor suspect;" about half of these will be rejected as negative on standard film examination; about 10 per cent of the "positive" standard films will yield verified bronchogenic carcinomas.

TABLE XI

Tumor Suspects on Survey Films ("over-reading")	Tumor Suspects on Standard Films	Bronchogenic Carcinomas Verified
1,000	500	50

In periodic surveys of mixed groups, male and female, symptomatic and asymptomatic, Boucot and Sokoloff[5] recently reported a yield of 210 tumor-suspect minifilms per 100,000 examined. The prevalence of bronchogenic carcinoma proved to be 37 per 100,000. Despite prompt hospitalization, the resectability rate in these was such that the three year survival rate was only about 9 per cent. In the patients without symptoms, none survived; in those with symptoms, a few survived—they presumably had slow growing or biologically favorable tumors.

The type and frequency of the survey procedure are variously recommended as follows:

Ochsner (1954): frontal film every 3 months on men over forty who have smoked at least one package of cigarettes daily for twenty years;

Blades (1954): frontal and lateral film every 6 months on adults;

Churchill (1953): frontal films (inspiration and expiration) and lateral every 6 months on males over forty;

Chocorua Lung Cancer Conference (1952): frontal films (inspiration and expiration) and lateral every 12 months on males over fifty.

It is now generally agreed that examination should be made at least every six months if it is to be at all effective.

As of July 1, 1954, there were approximately 22,000,000 males forty-five years of age and older in the United States. Examination of this group twice yearly on single films would necessitate about 44 million photofluorograms per annum being made, interpreted and filed. If three views were made at each visit, it would involve processing 132 million such films per year. Using standard size films for the follow-up of "tumor suspect" cases, about 1 million such exposures would be required annually. The clerical job alone of recording and following these cases would be rather considerable.

All workers are agreed that the problem of prompt *follow-up* and adequate treatment of the initial tumor suspects is one

of great magnitude. Despite extensive efforts, *less than 50 per cent* of the 3,500 tumor suspects in the Los Angeles survey were followed to definite diagnosis during the entire year after termination of the survey. Guiss attributes this to a combination of apathy or lack of understanding on the part of both physician and patient. He believes that the patients with lung cancers undoubtedly included in this group will probably have lost their chance of cure.

McNulty noted that in Boston, in addition to the above delay, the actual *resectability* rate of the promptly followed cases proved to be only about 50 per cent, indicating that "the survey did not uncover bronchogenic cancer in a significantly earlier phase than that seen in ordinary practice." The initial resectability rate in the Los Angeles survey was estimated as 64 per cent but with further experience it turned out to be only 39 per cent. Nevertheless, in analyzing several groups of surveys, we note a resectability rate of about 42 per cent compared with one of about 27 per cent in regular practice.

The short term "curability" of survey detected cancer is about 11 per cent to date. While slightly better than that of "regularly" diagnosed or symptomatic cancer, this rate is quite low, presumably because carcinoma of the lung is biologically an unfavorable type of tumor. Many patients with tumors of less than 2 cm. in diameter already have inoperable nodal or vascular metastases at time of exploration. While much can be done with radiotherapy to palliate these inoperable cases, most patients will not obtain significant increase in survival. Will we then be performing a useful service in disturbing with semiannual survey procedures this large group of persons to whom we have so little to offer in the way of true therapeutic benefit?

So much for primary lung cancer as a survey problem. It is realized that there is a useful by-product of chest surveys for bronchogenic carcinoma, that is the discovery of clinically important lesions of other nature. These include pulmonary adenomas, solitary tuberculomas and other granulomas, resectable solitary pulmonary metastases, and, of course, the various active inflammatory lesions such as tuberculosis.

The public health aspect of surveys is recognized in connection with such communicable disease. When a case of active pulmonary tuberculosis is detected and isolated, one source of spread is eliminated and thereby development of additional cases is prevented. However, when a case of pulmonary cancer is detected and treated the results are purely personal, and the community as a whole is not benefited or protected. Surveys for cancer are therefore not strictly public health measures.

SUMMARY

1. Photofluorographic screening procedures applied to the general population have led to the detection of about 10 cases of bronchogenic carcinoma per 100,000 persons examined.

2. In the surveys reported to date, about 8 per cent of shadows regarded as "tumor suspect" in films of asymptomatic adults proved to be bronchogenic carcinoma. About 24 per cent of "solitary circumscribed shadows" in such persons proved to be carcinoma.

3. If the procedures were restricted to males over forty-five, and conducted every six months, the yield should reach about *50 cases per 100,000* expecially if tested readers and dual readings are employed.

4. There are some 22 million males in this age group in the United States. Semiannual examinations, using three views (inspiration and expiration frontal and inspiration lateral) would involve some 132 million minifilms *annually*, plus the necessary million or so standard or other size films required for checking those with "tumor suspect" minifilms each year.

5. The psychologic problem of maintaining the continued interest of qualified readers in a program of this scope baffles

the imagination. It is not primarily a question of funds, energy, or medical manpower; it is a question of simple sustained interest involved in a study of asymptomatic persons when the yield, fortunately, is so small. Without qualified readers, the number of missed "positives" and the number of over-read "negatives" would probably defeat any wide-scale program.

6. In several recent large surveys analyzed in this paper, the prognosis in survey detected bronchogenic carcinoma proved little better than that of carcinoma detected by conventional methods of examining the chests of apparently symptomatic persons. This is not surprising in view of the biologic predeterminism of this particular type of neoplasm. Survey-detected lung cancer is unfortunately seldom "early."

7. Any effective screening procedure must have prior arrangements for prompt follow-up, completion of diagnosis and adequate treatment. Intervals of three to eighteen months tend to elapse between the making of the original tentative diagnosis and the actual therapeutic operation. Further, many asymptomatic persons refuse thoracotomy. The major portion of the potential benefit in discovering lung cancer by the screening procedure is therefore lost.

8. A screening procedure more effective than mass chest roentgenologic surveys is needed if a significant attack is to be made on the lung cancer problem. Pending the development of such it is probable that semi-annual roentgen examination of persons over forty-five (notably of heavy smokers?) could be recommended, especially is such persons are willing to submit promptly to the necessary surgical procedures in the event a shadow is discovered. However, the yield in lives actually saved or even made more comfortable by this program (in the light of known curative or palliative techniques) appears to be so small that one cannot urge its imposition on the general public. It would result in much apprehension on the part of the many "false" positives and, in all honesty, it would not save many lives.*

450 Sutter St.
San Francisco, 8, California

REFERENCES

1. Birkelo, C. C., and others. Tuberculosis case finding; comparison of effectiveness of various roentgenographic and photofluorographic methods. *J.A.M.A.*, 1947, *133*, 359–366.
2. Blades, B. B. Surgical management of tumors of lung discovered in x-ray surveys. *J.A.M.A.*, 1954, *154*, 196–198.
3. Bondi, G., and Leites, V. Malignant neoplastic disease discovered in chest x-ray surveys. *New England J. Med.*, 1952, *247*, 506–512.
4. Boucot, Katharine R., and Sokoloff, M. J. Preclinical bronchogenic carcinoma. *Am. Rev. Tuberc.*, 1954, *69*, 164–172.
5. Boucot, Katharine R., and Sokoloff, M. J. Is survey cancer of lung curable? J. Dis. Chest. To be published.
6. Chocorua Lung Conference, 1952. Recommendations of American Cancer Society, 47 Beaver St., New York, N. Y.
7. Churchill, A. S. Bronchogenic carcinoma in San Diego County. *California Med.*, 1953, *73*, 232–235.
8. Cochrane, A., and Garland, L. H. Observer error in interpretation of chest films; international investigation. *Lancet*, 1952, *2*, 505–509.
9. DeBakey, M. E., Ochsner, A., and DeCamp, P. T. Primary carcinoma of lung. *Surgery*, 1952, *32*, 877–891.
10. Denk, W. Bemerkungen zum Bronkuskarzinom. *Beitr. klin. Chir.*, 1953, *186*, 282–290.
11. Doll, R. In discussion of Korteweg, R. Cancer of lung in Netherlands. Louvain Symposium. Acta U.I.C.C., 1953.
12. Dorn, H. F. Morbidity and mortality from cancer of lung in U. S. Symposium on lung cancer, Louvain, Acta U.I.C.C., 1953.
13. Dowdy, A. Personal communication.
14. Garland, L. H. Scientific evaluation of diagnostic procedures. *Radiology*, 1949, *52*, 309–327.
15. Garland, L. H. On reliability of roentgen survey procedures. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 32–40.
16. Garland, L. H. Value of mass chest x-ray surveys in control of lung cancer. Stanford Med. Bull., 1953, *11*, 44–45.
17. Groth-Petersen, E., and Thillemann, J. On reliability of reading of photofluorograms and value of dual reading. *Acta tuberc. scandinav.*, 1952, *26*, 13–37.

* For discussion see page 436.

18. GUISS, L. W. Value of mass chest roentgen-ray survey methods in control of lung cancer. *Cancer*, 1952, *5*, 1035–1040.
19. GUISS, L. W. Dividends and defects of mass-survey method in detecting lung cancer. *Ca, Bull. Cancer Progress*, 1954, *4*, 56–59.
20. GUISS, L. W. Personal communication.
21. HEMLIN, L. E., Medical Director, American Brake Shoe Company. Personal communication, April 12, 1954.
22. HOOD, R. T., JR, GOOD, C. A., CLAGETT, O. T., and MCDONALD, J. R. Solitary circumscribed lesions of lung; study of 156 cases in which resection was performed. *J.A.M.A.*, 1953, *152*, 1185–1191.
23. HORN, D. American Cancer Society Bulletin, November, 1953, 521 W. 57 St., New York, N. Y.
24. JONES, J. C. Surgical aspects of bronchogenic carcinoma. *J.A.M.A.*, 1947, *134*, 113–117.
25. LOMBARD, H. L., MIDDLETON, M., WARREN, S., and GATES, O. Use of vaginal smear as screening test. *New England J. Med.*, 1952, *246*, 523–528.
26. MCNULTY, J. M. Clinical follow up study of 398 patients suspected of having lung cancer discovered in Boston Chest X-Ray Survey. *New England J. Med.*, 1954, *250*, 14–17.
27. MAY, I. A., ROSE, K., and DUGAN, D. G. Solitary pulmonary lesion. *California Med.*, 1954, *80*, 9–12.
28. MOORE, R. L. Primary cancer of lung. *Cancer*, 1951, *4*, 663–668.
29. OCHSNER, A., DECAMP, P. T., and RAY, C. J. Bronchogenic carcinoma with emphasis on early diagnosis. *Geriatrics*, 1954, *9*, 15–19.
30. OVERHOLT, R. H. Cancer detected in surveys. *Am. Rev. Tuberc.*, 1950, *62*, 491–500.
31. OVERHOLT, R. H., and ATWELL, S. W. Carcinoma of the Lung. American Cancer Society, New York, 1950.
32. OVERHOLT, R. H. Personal communication, July 17, 1954.
33. SCAMMAN, C. L. Follow-up study of lung-cancer suspects in mass chest x-ray survey. *New England J. Med.*, 1951, *244*, 541–544.
34. STATISTICAL BULLETIN, Metropolitan Life Insurance Co., February, 1953.
35. STEINER, P. Cancer of lung. Louvain Symposium. Acta U.I.C.C., 1953.
36. THERKELSEN, E., and SØRENSEN, H. R. Diagnosis, determination of operability and differential diagnosis in bronchogenic carcinoma. *Acta chir. scandinav.*, 1953, *106*, 1–23.
37. VITAL STATISTICS OF THE U. S. Vol. III. U. S. Dept. H. E. W., 1950.
38. YERUSHALMY, J., HARKNESS, J. T., COPE, J. H., and KENNEDY, B. R. Role of dual reading in mass radiography. *Am. Rev. Tuberc.*, 1950, *61*, 443–464.

Vol. 74, No. 3

THE ROENTGEN SIGNS OF CARCINOMA OF THE LUNG*

By LEO G. RIGLER, M.D.
MINNEAPOLIS, MINNESOTA

INTRODUCTION

THE problems presented by the increasing frequency with which cancer of the lung is encountered have been thoroughly explored on many occasions. The virtues of roentgen examination as a method of detection are generally accepted. Certainly there is no other means so generally applicable whereby tumors of the lung can be found at an earlier stage. Many survey studies have established that simple posteroanterior and lateral roentgenograms of the chest will permit the detection of an abnormality in the lung, which may later be identified as a carcinomatous process, long before the onset of symptoms.

While the detection of an abnormal process in the lungs can be accomplished with relative ease and accuracy, the identification of the nature of the disease presents many more difficulties. This is especially true of carcinoma of the lung for it represents the great simulator of all other types of disease of the lungs. A pathological state which may produce a growing mass in the lung, necrosis and abscess formation, secondary inflammatory reaction, perilymphatic and perivascular infiltrative processes, partial or complete bronchial obstruction, segmental or otherwise, with all the pulmonary sequelae which follow, obviously may simulate almost any of the lesions to which the lung is heir.

With the increasing use of roentgen examination in apparently normal individuals the problem of differential diagnosis has become an extremely pressing one. The finding of an abnormal shadow in the roentgenogram of the lungs of an apparently well individual puts a grave responsibility upon the physician in charge. There are many methods whereby a definitive diagnosis of carcinoma of the lung may be established. The most cogent of these, of course, is the microscopic examination of tissue removed either through the bronchoscope, by percutaneous biopsy, or as a result of thoracotomy. Direct visualization of the tumor through the bronchoscope is an extremely effective and often practically definitive method. The demonstration of carcinomatous cells in the sputum or in bronchial aspirates is another effective method for the positive identification of lung cancer. Yet there are still a substantial number of patients with an abnormal process in the lung, in whom none of these methods is effective. In some instances it is because of inability or reluctance to apply such methods. For example, percutaneous lung biopsy is not widely accepted as a reasonably safe procedure. Despite the easy assurance of thoracic surgeons and despite the relatively innocuous character of exploratory thoracotomy, there are many cogent reasons why certain patients cannot be submitted to this procedure. Bronchoscopy, either by direct visualization or by the use of the biopsy, has many limitations. At the present time less than 50 per cent of all the patients operated upon for carcinoma of the lung have had either a positive biopsy or positive bronchoscopic visualization of the tumor. This is, of course, particularly true of the peripheral spheroidal nodules which are now so commonly being found. The demonstration of cancer cells in the sputum is of great value when positive but there are many cases in which the method is ineffective either because of lack of spu-

* From the Department of Radiology, University of Minnesota Medical School, Minneapolis, Minnesota. Presented in a Symposium on Carcinoma of the Lung at the Fifty-fifth Annual meeting, American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

tum or failure to shed cells. Furthermore, it is a method which has been successful only in a relatively small number of institutions and is not widely applicable.

It is usually held that while roentgen examination may be of great value as a means of detection its utility as a method of identification is sharply limited. That there are distinct limitations in the roentgen determination of the exact nature of a disease process, no one can gainsay. But there are distinctive roentgenologic signs which seem so characteristic of malignant tumors that they carry with them almost the same weight as the more positive finding of actual cells, whether in the biposy specimen or in the sputum. In this connection I would emphasize that all diagnoses, including those derived from autopsy findings, are only probabilities. Certainly errors occur in the interpretation of the bronchoscopic biopsy, in the bronchoscopic observations of tumor, even in biopsies obtained from exploratory thoracotomy. The following case is illustrative.

CASE REPORT

A male, aged sixty-one, came in with a history of acute pain in the chest, cough and distress beginning about three weeks before. His physician thought he had a virus pneumonia and put him to bed for a time. He was given some medicine which caused him to evacuate a great deal of sputum, following which he felt much better.

The patient had a history of cough with hemoptysis since the age of fifteen occurring at infrequent intervals. During the past few years he had had several attacks of such coughing spells but without hemoptysis.

Rogentgen examination of the chest was done and his physician, suspecting a lung tumor, sent him in for examination.

On entrance the temperature was 100.4° F., the leukocyte count 14,500, with 72 per cent neutrophils. Laboratory findings were otherwise negative. Vital capacity was reduced, being 67 per cent of normal. Repeated examinations of the sputum showed no evidence of bacteria. A specific study for tumor cells was made with negative results.

Roentgen examination of the chest (Fig. 1*a*) revealed a consolidating process in the right upper lobe involving essentially the anterior axillary segment with some extension, however, into the apical portion. The posterior segment appeared to be clear. The area of density was notable by reason of the bulging character of a mass which extended somewhat inferiorly and anteriorly. The diaphragm was not especially elevated and there were no other evidences of atelectasis. The appearance suggested a carcinoma of the anterior axillary branch of the right upper lobe with tumor extension into the parenchyma of the lung and partial atelectasis. Laminagraphic studies, both anteroposterior and lateral (Fig. 1, *b* and *c*) were made and revealed an obstruction of the axillary branch of the right upper lobe bronchus just as it approached the mass. Some evidence of necrosis within the mass could also be made out. There was also some evidence of enlarged lymph nodes in the hilus. Comparison was made with films made by his local physician three weeks earlier, showing no change.

Because of the finding of stenosis in the laminagraphic study and the bulging mass, a diagnosis of carcinoma of the anterior axillary branch of the right upper lobe was made.

The location of the lesion was such as to indicate that bronchoscopy would not be highly successful since the bronchus was open for several centimeters beyond the origin of the upper lobe bronchus.

Exploratory thoracotomy was undertaken by Dr. Richard Varco. The pleura exhibited plaques of thick, whitish tissue. Biopsies made from such areas, however, indicated that it was purely a fibrous tissue reaction and no tumor could be made out. An extensive nodular lesion could be palpated in the anterior segment of the right upper lobe and enlarged lymph nodes in the mediastinum could also be felt. Sections were made from the parenchyma of the lung and from the lymph nodes by the frozen tissue technique and the pathologist reported that this was entirely a chronic inflammatory disease. Despite this, Dr. Varco felt that pneumonectomy was indicated because of the characteristic roentgen signs and the clinical evidences to suggest carcinoma, and a right pneumonectomy was done.

The specimen revealed a palpable lesion in the upper lobe measuring approximately 4.5 cm. in diameter. This extended well out to the pleura. The tumor mass originates in the an-

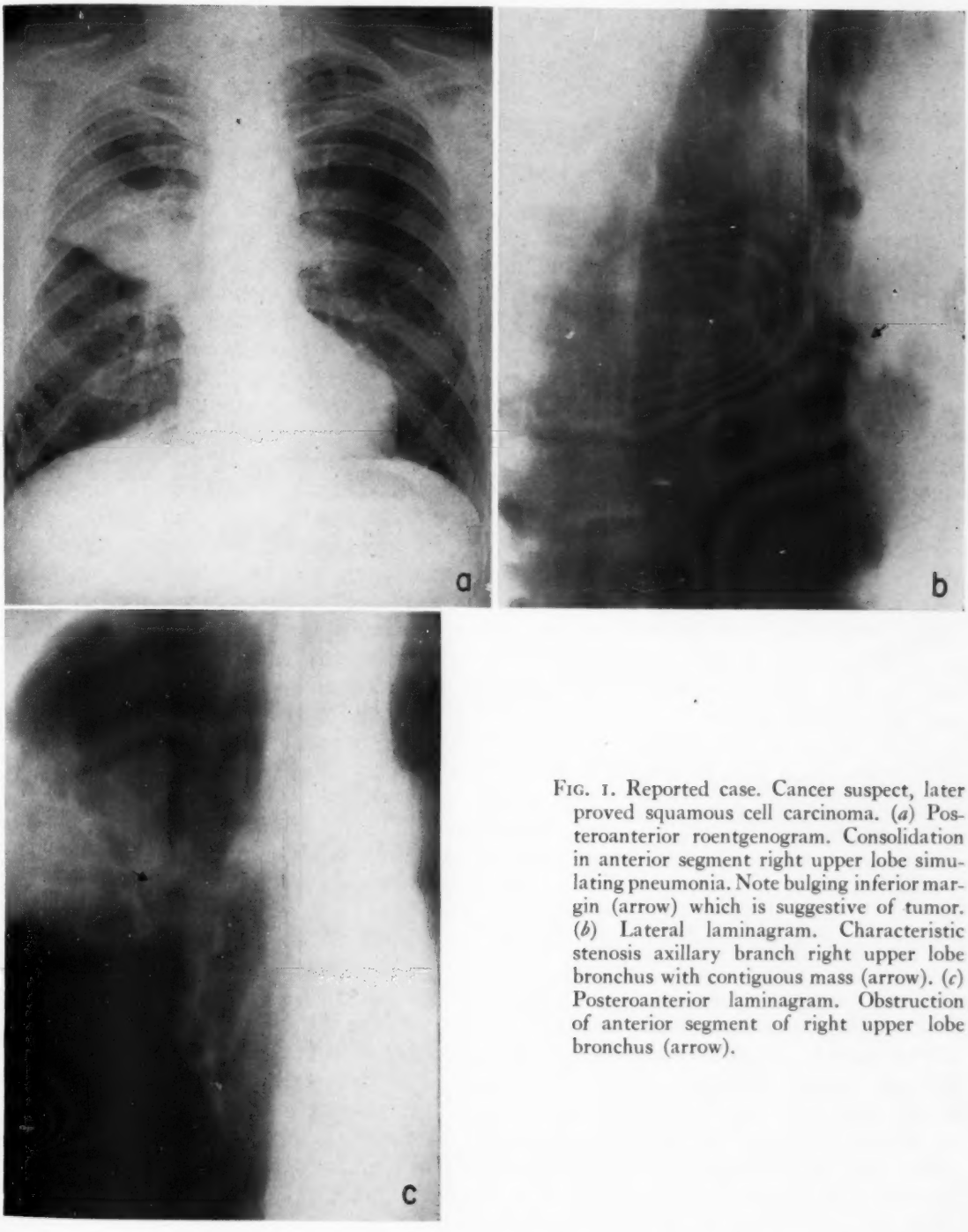

Fig. 1. Reported case. Cancer suspect, later proved squamous cell carcinoma. (*a*) Posteroanterior roentgenogram. Consolidation in anterior segment right upper lobe simulating pneumonia. Note bulging inferior margin (arrow) which is suggestive of tumor. (*b*) Lateral laminagram. Characteristic stenosis axillary branch right upper lobe bronchus with contiguous mass (arrow). (*c*) Posteroanterior laminagram. Obstruction of anterior segment of right upper lobe bronchus (arrow).

terior segment of the upper lobe bronchus. This bronchus is occluded and disappears into a small cavity measuring approximately 1 cm. The bronchus shows a ragged lining as does the cavity. The surrounding mass is composed of compressed and infiltrated lung tissue containing pockets of purulent material. Multiple enlarged lymph nodes are found in the hilus. The main upper lobe bronchus and the posterior and axillary branches appear to be normal.

Microscopic sections through the occluded end of the involved bronchus gave a charac-

teristic picture of squamous cell carcinoma. Sections of fifteen lymph nodes show four of these nodes to contain tumor similar to that found in the lung. No tumor was found in the pleura on restudy.

DISCUSSION

In this case both the examination of the sputum and biopsies taken from the parenchyma of the lung and from the lymph node and pleura at operation, as done by the frozen tissue technique, failed to show tumor.

The roentgen findings of a chronic infiltrative process in the lobe with cavitation plus an expanding outer margin, together with a fairly characteristic stenosis of the bronchus were indicative of carcinoma. The added finding of lymph nodes in the hilus gave further confidence in this diagnosis.

Obviously, such a failure of pathology and success of roentgenology is the exception but the case is cited to indicate that in all diagnoses there is the possibility of error and one must weigh all the evidence before coming to a conclusion.

There are many patients with demonstrable lung pathology in whom a definitive diagnosis appears impossible regardless of what method may be used, and exploratory thoracotomy seems to be the only feasible procedure. But surgical invasion of the thorax is not to be undertaken lightly, regardless of the facility with which it may now be accomplished. For one thing it is often difficult to persuade an individual who appears to be perfectly healthy to accept an exploratory thoracotomy only because, during the course of a survey examination, he has been found to have an abnormal shadow in the lung. Everyone has experienced failures in this regard. In many other cases there is a great reluctance on the part of physicians to recommend a procedure of this order when the evidences in favor of a malignant lesion are so minimal.

The problem has become particularly acute at this time, especially because of the solitary spheroidal nodule of the lung. More and more, as patients have routine examinations of the chest, these solitary pulmonary shadows come to plague us and demand some procedure which will make it possible to determine their exact diagnosis. The same is true of the minor pulmonary infiltrations which we are encountering in apparently normal individuals, infiltrations which we know may be manifestations of carcinoma.

Intensive roentgenologic study should be undertaken in such cases in order to determine the presence or absence of certain roentgen signs which have great diagnostic significance. In order to observe the findings described below it is necessary to make conventional roentgenograms in a variety of positions, to do laminagraphy and, if necessary, bronchography.

Of great importance is the comparison of films made at various times in the history of the patient. A very high percentage of our adult population at the present time will give a history of having had roentgenographic examination of the chest, often fortuitously, at some previous date. Whether done by the photofluorographic method or by the use of full-sized films, such records should be preserved for many years. This is especially true of what were thought to be negative roentgenograms, for often such films actually exhibit the evidences of a very early lesion, not sufficiently apparent to be detectable at the time, but easily seen in retrospect when a larger lesion is found later.

None of the roentgen signs to be described is absolutely diagnostic but the positive findings have a degree of probability which approaches that achieved by microscopic studies. Furthermore, their reliability is sufficient to present a firm indication for exploratory thoracotomy.

DEFINITIVE ROENTGEN SIGNS OF CARCINOMA OF THE LUNG

1. The appearance of a solitary pulmonary shadow not present in previous films, especially in an individual past middle age, is of very great significance. It is obvious at

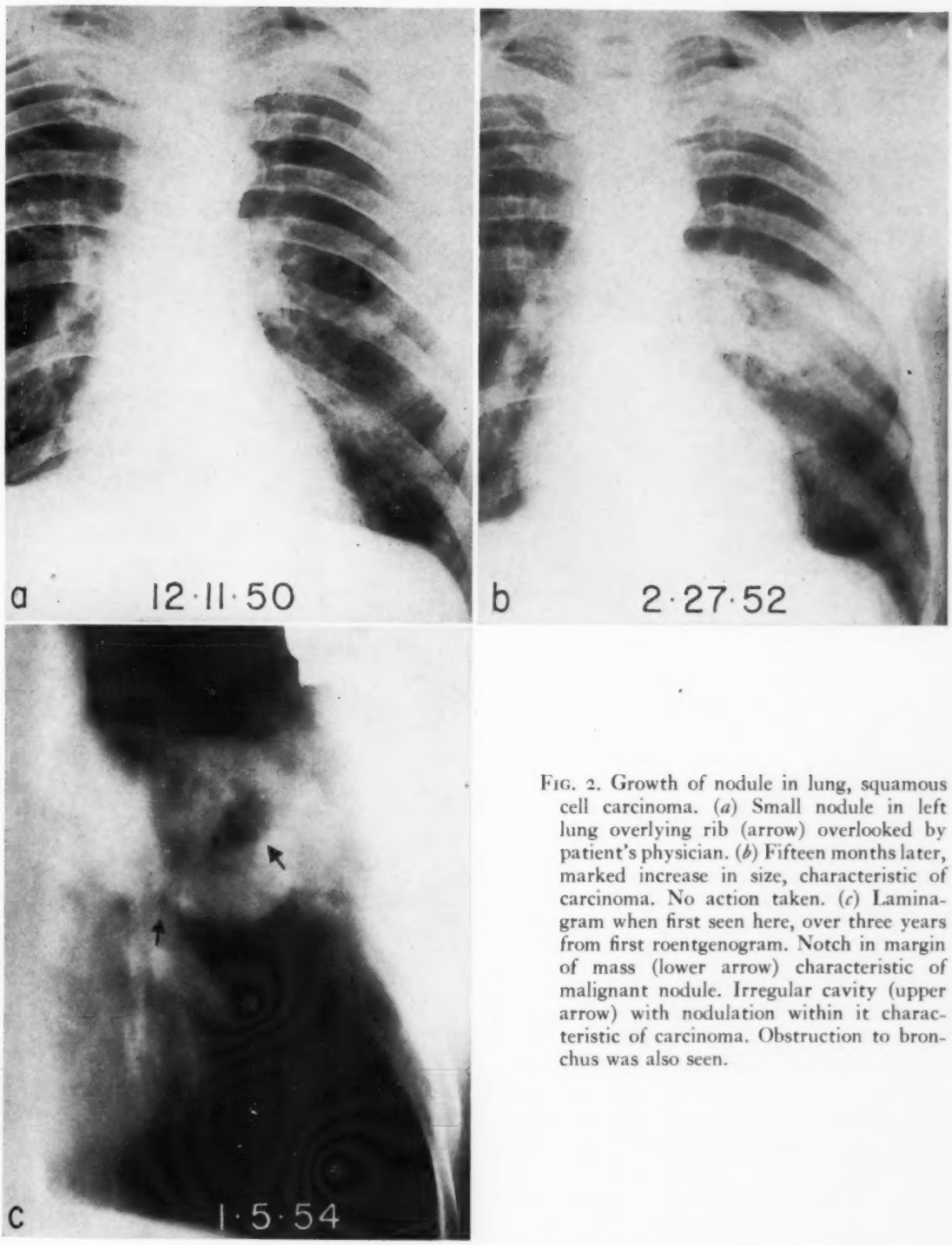

Fig. 2. Growth of nodule in lung, squamous cell carcinoma. (*a*) Small nodule in left lung overlying rib (arrow) overlooked by patient's physician. (*b*) Fifteen months later, marked increase in size, characteristic of carcinoma. No action taken. (*c*) Laminagram when first seen here, over three years from first roentgenogram. Notch in margin of mass (lower arrow) characteristic of malignant nodule. Irregular cavity (upper arrow) with nodulation within it characteristic of carcinoma. Obstruction to bronchus was also seen.

once that here there must have been a previous examination at a period of time usually longer than three months before the one under consideration. Almost invariably such a situation is encountered in individuals who are completely symptomless. The complete absence of any evidence of a lesion in an area which in later films

shows infiltration or a nodule is a specific indication for resection of the area with more radical procedure if the diagnosis of carcinoma is confirmed microscopically at the time of operation.

The demonstration, in an individual over forty, of a nodular pulmonary shadow which was not present in films made six months, a year, or several years before is not by any means complete assurance that the lesion is a tumor or that it is malignant. We have learned in recent years, from bitter experience, that granulomas will likewise appear suddenly in middle-aged individuals. But I believe this is, by far, the exception and not the rule. A lesion of this kind appearing suddenly is much more likely to be a carcinoma than it is to be a benign process or a granuloma.

2. The demonstration of a peripheral pulmonary mass or nodule in the lung which shows an increase in size when compared to a previous film is likewise of very great importance (Fig. 2, *a* and *b*). This is perhaps an even more cogent indication of the presence of a malignancy than the sudden appearance of a shadow in an area in which none was previously seen. But, again, there are exceptions since granulomas may increase in size under observation, as we have seen, on many occasions. It should be noted, in this connection, that a failure to increase in size does not at all exclude carcinoma. Some malignant tumors grow very slowly. I have observed cases of carcinoma which took as long as seven years to attain a considerable size. Furthermore, I have observed at least one case in which over a period of four years there was no change whatever in size, although it proved to be an adenocarcinoma of the lung. Despite these rather startling exceptions, the probability that a lesion which has increased in size over a period of one year, especially if the increase is reasonably rapid, is a carcinoma is extremely high and should certainly so be regarded until proved otherwise.

3. In any solitary peripheral pulmonary nodule the possibilities of diagnosis are always very difficult. Some thirty conditions may produce such a shadow in the roentgenogram. Laminagraphic studies, however, may be of very great value in determining whether or not the lesion is malignant or benign. By this means an accurate determination as to the sharpness of outline, the homogeneity, and the contour of the lesion can be obtained. None of these characteristics are highly diagnostic, however, since granulomas may be quite sharply rounded while tumors may or may not be sharply defined. The homogeneity, likewise, is not distinctive since cavitation or necrosis (Fig. 2*c*) may occur both with tumors and with inflammatory processes. The contour may be of some importance because most tumors tend to be reasonably regular in their outline and to assume a spheroidal shape, whereas inflammatory lesions may be much more irregular and much less geometric in form.

Of greater importance is the demonstration of the presence or absence of calcium in the mass. Such a determination is best made by laminagraphy; the procedure is highly effective in demonstrating calcium. While it is true that the absence of calcium is of little significance, since many granulomas and benign lesions do not show calcium, the presence of calcium is of great significance. A few cases, both of bronchial adenoma and of carcinoma, have been reported in which calcium was demonstrated in the lesion. In some of these the calcium may well have been present previous to the development of the tumor which happened to occur in the same area. In others, necrosis of the tumor resulted in calcification. Nevertheless, these are startling and extraordinary exceptions to the general rule. In my own experience I have never seen a true carcinoma of the lung with calcification, and this is the general opinion of many men who have had long experience with these tumors. For practical purposes, therefore, the presence of calcification, barring compelling evidence to the contrary, should indicate that the lesion is not a malignant tumor.

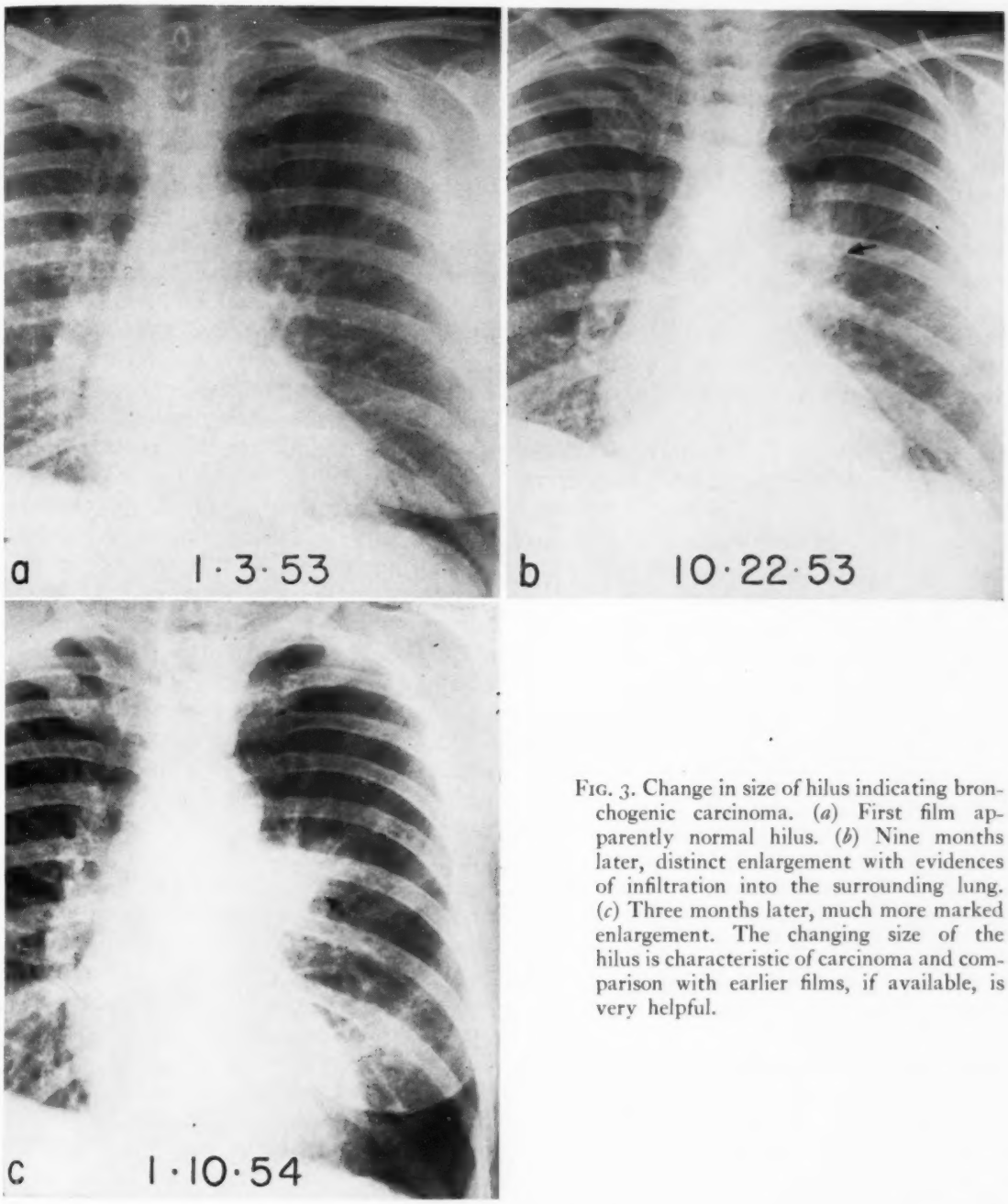

Fig. 3. Change in size of hilus indicating bronchogenic carcinoma. (*a*) First film apparently normal hilus. (*b*) Nine months later, distinct enlargement with evidences of infiltration into the surrounding lung. (*c*) Three months later, much more marked enlargement. The changing size of the hilus is characteristic of carcinoma and comparison with earlier films, if available, is very helpful.

4. In the course of our study of a great many nodules in the lung we have come upon a sign which appears, so far, at least, to be characteristic of malignancy. This consists of a notching or umbilication in the margin of the shadow (Fig. 2*c*). The deformity is not well seen in the ordinary roentgenograms, although occasionally we have seen it here as well, but it is quite clearly delineated in body section roentgenograms when the film is made at the proper level. The cause of this notching is not entirely clear although in several sections, which we have seen histologically, it

appears to resemble a hilus in the mass, containing blood vessels and normal lung structures extending into the body of the mass itself, thus producing the irregularity of the contour. The notching is usually single although in some cases it has been multiple. I have observed this in pulmonary metastases, such as from hypernephroma or melanoma, as well as in primary malignancies. Assuming that a solitary metastasis is an indication for extirpation, the presence of this notch seems to be a prime indication for immediate surgery with extirpation of the lesion.

It should be noted that the absence of this notch does not by any means exclude malignancy. While we have found it in small as well as large lesions, it appears more obvious in the smaller lesions. We have, however, seen it in masses as large as 10 cm. in diameter; we have seen it in lesions as small as 1 cm. in diameter. I would emphasize that the presence of such a notch, as seen in laminagraphic studies, is of utmost importance in determining that the patient has an actual malignancy. As yet we have not seen it in any benign condition.

5. A change in the size and appearance of one hilus is a lesion likewise of very great significance and only possible to observe when previous films are available (Fig. 3). Changes in the size of the hilus occur in probably 50 per cent of all the carcinomas of the lung. They are difficult to interpret because of the common occurrence of irregularities in the hilus resulting from inflammatory changes and other disease processes in the lung. Nevertheless, an enlargement of one hilus, even by itself, should be considered a carcinoma until proved otherwise. If the hilus seems to have been enlarging while under observation the probability that it is a carcinoma is extremely high, much higher in fact than if one sees a peripheral lesion enlarging.

It is at once apparent that the demonstration of an enlarged hilus or even the suspicion of enlargement of the hilus should call for intensive roentgen study. Several positions including oblique and lateral views (Fig. 4*c*) should be made. Such films will often permit a more definite determination as to whether the shadow seen is truly in the hilus and not in the parenchyma of the lung superimposed upon it, and whether an actual enlargement is present. Under such circumstances, roentgenograms made in inspiration and expiration may be of very great value. For a hilar enlargement of carcinomatous origin is usually the result of a tumor arising in one of the major bronchi which has extended into the peribronchial tissues. Such tumors very often produce sufficient obstruction of the bronchus to exhibit an obstructive emphysema (Fig. 4*b*) in the area supplied by the involved bronchus. It is best brought out in expiration; this is an extremely important maneuver in determining definitely whether an actual tumor is present (Fig. 4, *a* and *b*). Further steps should include laminagraphy, in an effort to visualize the major bronchi, since an enlargement of the hilus is almost always associated with a tumor arising in a bronchus of the first, second, or third order (Fig. 4*d*). Furthermore, the presence of enlarged lymph nodes can be effectively determined in this fashion. If the laminagraphic study is not entirely satisfactory, that is if the major bronchi are not clearly visualized throughout, it may be necessary to go a step further and do bronchography with an opaque medium. The latter should lead finally to a definitive determination as to whether or not any partial or complete obstruction of a bronchus is present and the nature of that obstruction. The latter will be considered later.

6. An infiltrative lesion in the lung is perhaps the most difficult of all to differentiate, particularly from the various forms of pneumonitis and from tuberculosis. There are two roentgen signs which are of some value in this differentiation. The first is, again, the demonstration over a period of time of the increasing character of the infiltrative process. While it is true

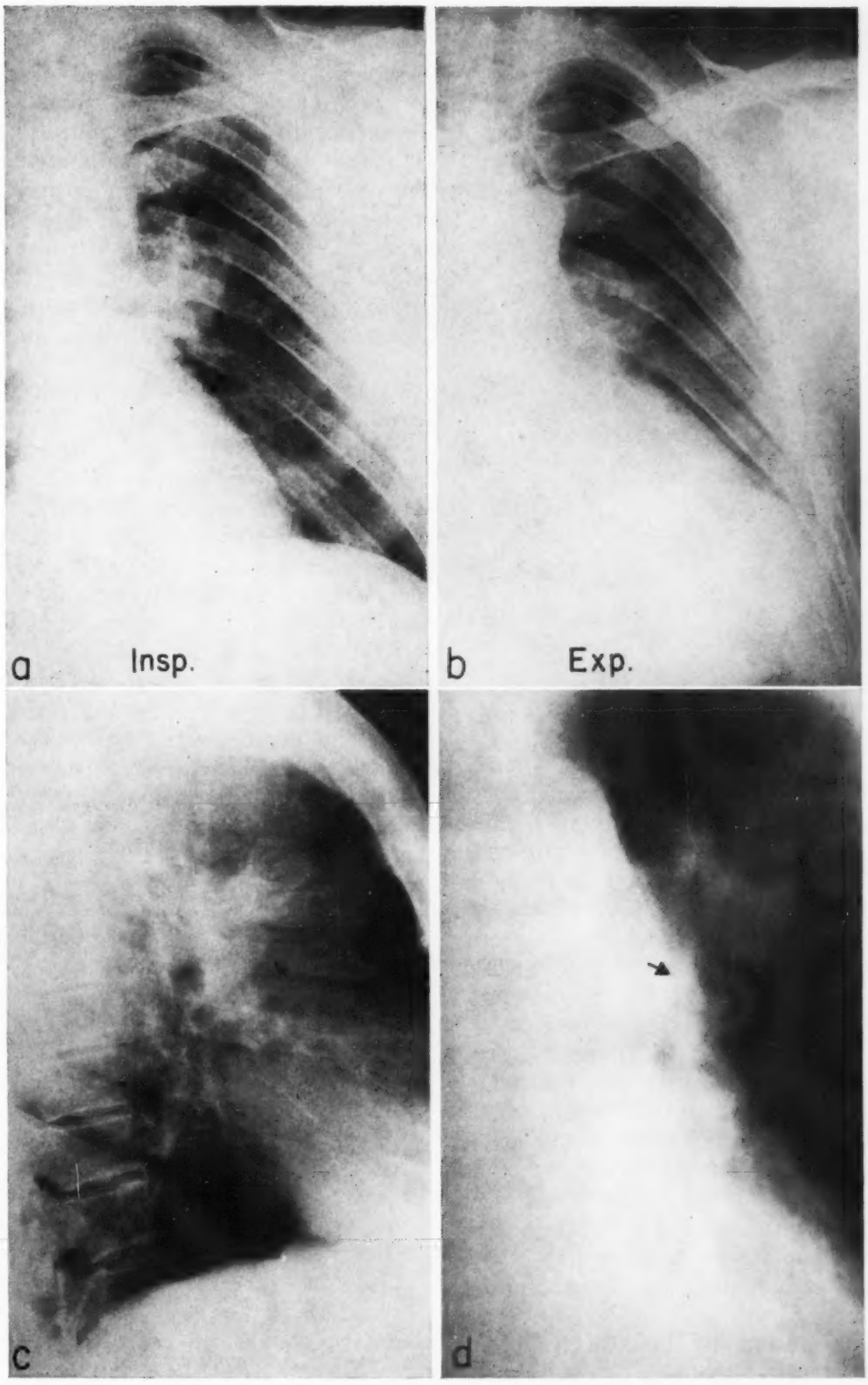

FIG. 4. Carcinoma of lung exhibiting enlarged hilus and obstructive emphysema. (*a*) Inspiratory roentgenogram. Markedly enlarged left hilus but with apparently normal appearing lung. (*b*) Expiratory roentgenogram showing marked radiability of upper lobe characteristic of obstructive emphysema. (*c*) Lateral view indicating that mass is actually in hilus but with some extension into upper lobe. (*d*) Laminagram showing tumor mass projecting into upper lobe bronchus (arrow) which tends to clinch the diagnosis.

Fig. 5. Posteroanterior roentgenogram in an infiltrating carcinoma of the lung. The nodular character of the infiltration is well shown (arrows). Such rounded, beaded areas along the course of an infiltration usually indicate its carcinomatous origin.

that this may occur in tuberculosis and other chronic lesions, usually it is much slower and less obvious, while in carcinoma the infiltrative process may gradually, but usually much more rapidly, increase in size and in extent of involvement. Secondly in such infiltrative lesions, a nodular beaded character can be demonstrated which is highly significant (Fig. 5). I would say at once that the definitive character of such lesions is far from that which I have discussed under the peripheral lesions or the enlarged hilus. Nevertheless, an infiltrative lesion which exhibits these nodulations along its course should certainly be considered highly suspicious, at least, of malignancy, despite its resemblance to an inflammatory process in other respects.

7. The pneumonic lesions are among those which give us the greatest difficulty in differential diagnosis. There are three roentgenologic findings which should arouse suspicion. (1) The failure of an ordinary pneumonia to resolve should be regarded as indicative of either carcinoma or a chronic process until proved otherwise. The diagnosis of unresolved pneumonia undoubtedly is correct is some cases but it is a highly risky diagnosis and should never be made without the most careful and repeated observation with roentgen examination. (2) The laminagraphic studies of such areas of infiltration of the lung, which are thought to be pneumonia, may again reveal a nodular type of mass previously indicated. This should suggest its carcinomatous origin. Furthermore, a typical bronchostenosis may be demonstrated in this way (Fig. 6). (3) Bronchography should be undertaken if the bronchi are not clearly delineated in the laminagram for very often in carcinomatous lesions which imitate pneumonia or have associated pneumonitis bronchostenosis can be demonstrated. Bronchostenosis does not accompany pneumonitis; it seldom occurs with chronic tuberculosis of the infitrating variety. The demonstration, therefore, of a bronchial occulsion, particularly if it is ragged and irregular, should lead to the diagnosis of carcinoma or at least should be sufficiently compelling to indicate the need

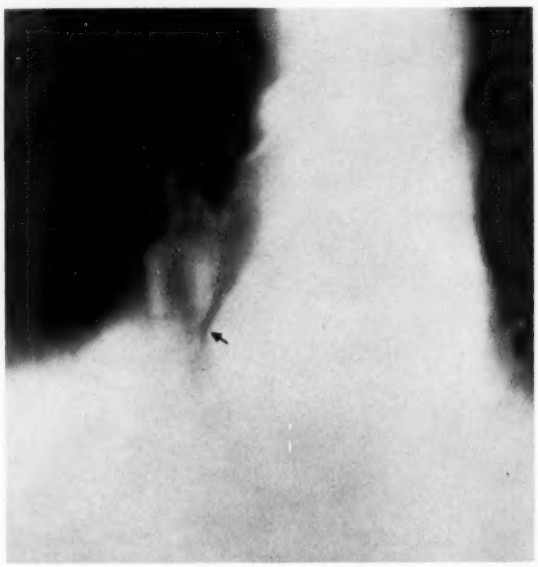

Fig. 6. Posteroanterior laminagram in a case of carcinoma of the lower lobe. Characteristic "rat-tail" obstruction of the lower lobe bronchus is well demonstrated (arrow).

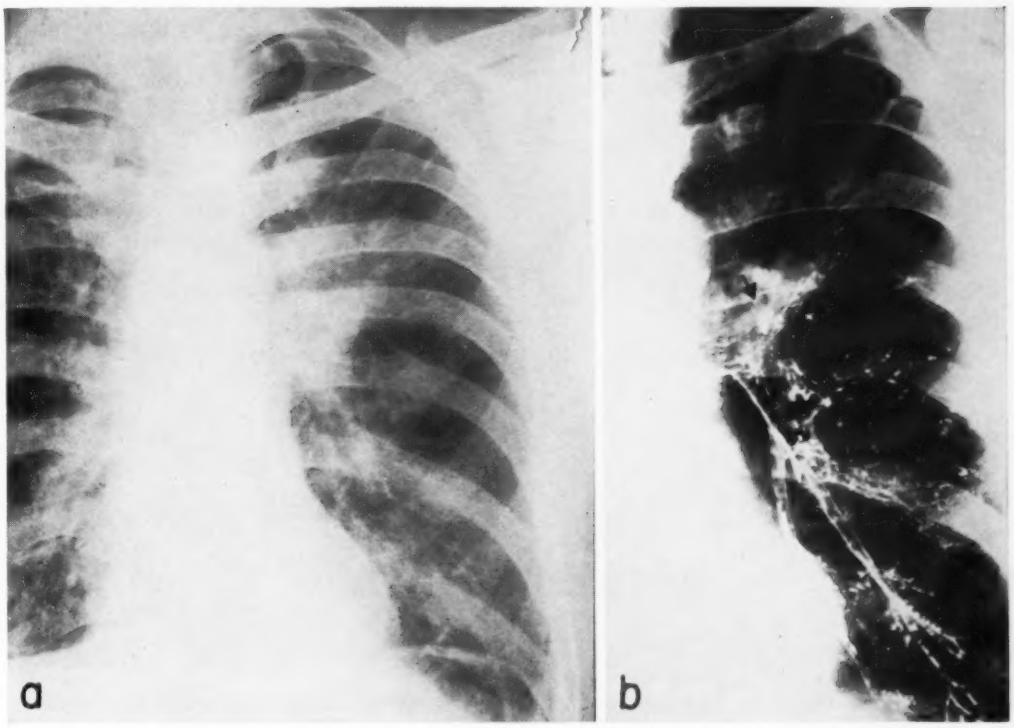

Fig. 7. Case of carcinoma, symptoms and roentgen signs simulating pneumonia. (*a*) Posteroanterior roentgenogram with enlargement of hilus and infiltration in the lung difficult to distinguish from unresolved pneumonia. (*b*) Bronchogram showing characteristic obstruction of the apical branch of the left upper lobe bronchus (arrow). This is typical of carcinoma.

for exploration (Fig. 7). A diagnosis of unresolved pneumonia should not be made without undertaking laminagraphic or bronchographic demonstration of the bronchi.

The contrast between an enlarged hilus with pulmonary infiltration of carcinomatous origin and that of inflammatory origin is demonstrated by Figures 7 and 8. In the latter, a case of unresolved pneumonia, the bronchi are seen to be normal.

8. A solitary abscess, even rather small in size, may be the first indication of a carcinoma of the lung. Such abscesses are usually the result of necrosis within a rapidly growing tumor. The distinction from an ordinary pulmonary abscess or from a tuberculous cavity can very commonly be made, especially if laminagraphy is undertaken. Carcinomatous abscesses will usually show a protruding outer margin which extends well beyond the area of the cavity itself (Fig. 9). The appearance of this protrusion is characteristic of a growing mass. In my own experience it has been present almost invariably in the carcinomatous abscesses, especially if one obtained good laminagraphic studies so that the clear delineation of the outline of the borders of the cavity could be made. In the larger cavities the presence of masses within the air pocket itself can be made out, indicating tumor nodules growing within the cavity (Fig. 2*c*). It is sometimes difficult to distinguish such shadows from those produced by granulations but, as a matter of fact, it is uncommon for a nontuberculous abscess or a tuberculous abscess cavity to show such masses within the air space.

9. The presence of segmental, lobar or unilateral emphysema, especially in the expiratory phase, is indicative of partial bronchostenosis. This, of course, is far from definitive insofar as carcinoma is

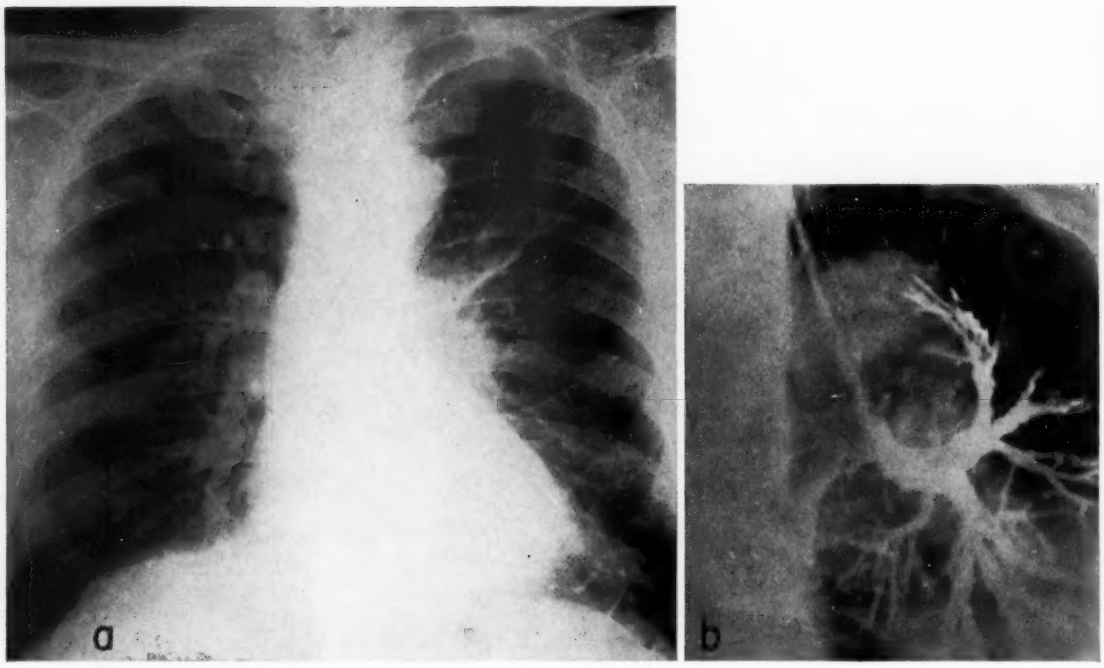

FIG. 8. Case of unresolved pneumonia, symptoms and signs simulating carcinoma. (*a*) Posteroanterior roentgenogram showing enlarged hilus with infiltration into the lung similar to Figure 7. (*b*) Bronchogram showing all branches of the left bronchus completely filled out and intact except for slight distortion due to the scar formation in the lung. The value of bronchography in such cases is illustrated.

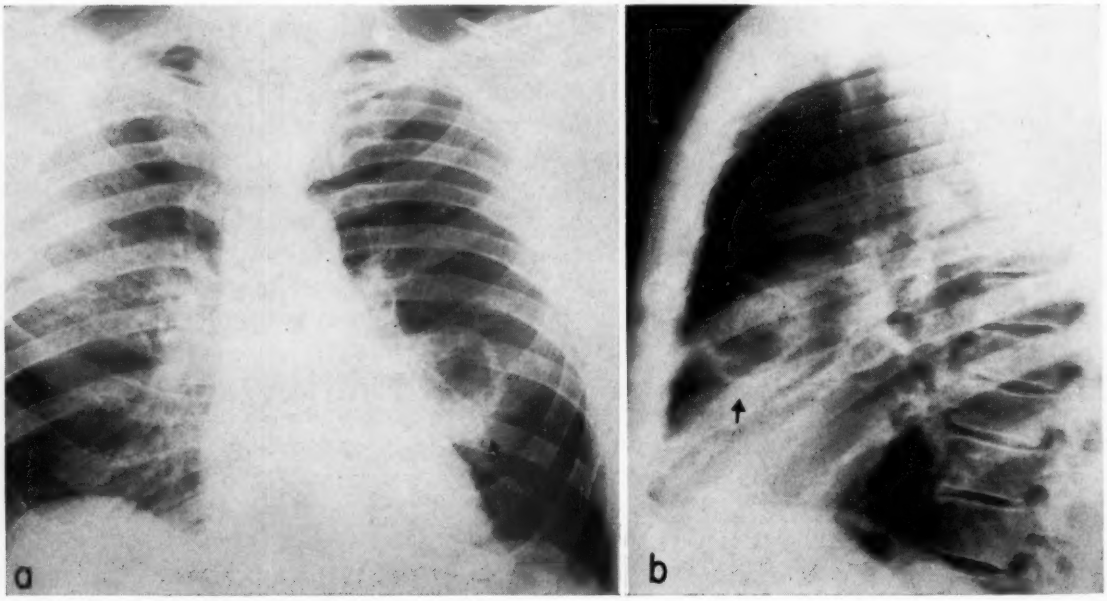

FIG. 9. Pulmonary abscess of carcinomatous origin. (*a*) Posteroanterior view. Characteristic cavity with fairly thick wall with little fluid within it. Note thick wall at inferior portion (arrow). (*b*) Lateral view. The bulging portion of the abscess, which is characteristic of carcinoma, is shown in the inferior portion (arrow).

concerned but if found in an individual past middle age it should certainly arouse first of all the thought that this is carcinoma. Further investigation, therefore, with laminagraphy or bronchography to determine the nature of the bronchostenosis is indicated. Very often, as will be observed later, the character of the occlusion thus observed may indicate clearly the fact that this is due to a carcinoma of the bronchus. The combination of emphysema and of an enlarged hilus almost invariably connotes carcinoma (Fig. 4).

10. The same indications are present in atelectasis as in emphysema. Obviously, atelectasis may be the result of a benign type of bronchostenosis but, again, in an individual past middle age in whom it has suddenly appeared the suspicion that this is due to a carcinomatous obstruction of the bronchus is a first consideration. Here, again, laminagraphy or bronchography may exhibit effectively the nature of the bronchostenosis and thus establish the diagnosis.

11. The demonstration of changes in the bronchial lumen can be made effectively in most cases by laminagraphy. Both posteroanterior and lateral laminagrams should be made and the technique is of great importance. When satisfactory, it is possible to delineate the bronchi of the first, second and third orders quite effectively. Smaller bronchi are not well demonstrated by laminagraphy. The demonstration by means of laminagraphy of a tumor as a positive shadow within the bronchus is not rare in carcinoma and may be a crucial deciding point in determining the presence of the tumor. It may be difficult to distinguish between benign adenoma and a malignant tumor on this basis, but at any rate a sufficient indication of the neoplastic nature of the process is afforded to make surgery imperative. A cap shaped indentation of the bronchial lumen either at its end or on one margin is also characteristic. Finally, the demonstration of an elongated, somewhat irregular stenosis of the bronchus is indicative of malignant obstruction (Fig. 1*c*, 2*c*, 4*d* and 6). In doing laminagraphy it is of great importance to get mutliple sections at various levels to be certain that the stenosis is not being simulated by an angulation of the bronchus. With these provisions in mind, however, it may be possible to demonstrate relatively small lesions in this fashion.

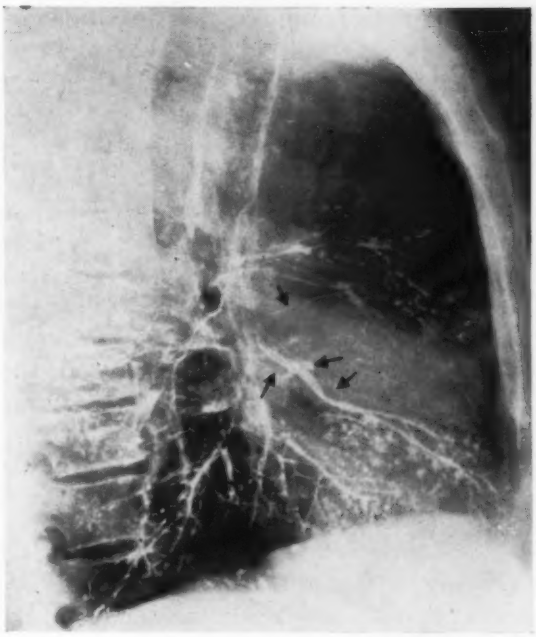

Fig. 10. Carcinoma of segment of right middle lobe difficult to diagnose by most procedures. Bronchogram, lateral view, shows characteristic obstruction of superior branch of right middle lobe bronchus with deformity and compression of inferior branch from the tumor itself (arrows). The other arrows indicate the interlobar fissures outlining the middle lobe, which is completely dense.

In the smaller bronchi and occasionally, because of technical difficulties, in the major bronchi, laminagraphy may be unsuccessful. Under these circumstances the introduction of a contrast medium into the bronchi through a catheter localized to the area under suspicion may be a final method of definitive diagnosis, assuming that bronchoscopy has been unsuccessful in this regard (Fig. 7*b* and 10). Small branch bronchi may be delineated and the obstruction within them determined. The character of the obstruction, whether exhibited

by means of laminagraphy or bronchography, is reasonably definitive for carcinoma of the bronchus. Such findings are of particular importance in the cases with apparent inflammatory infiltrates, unresolved pneumonias, etc., which are so often actually due to carcinoma and in which so much delay so frequently occurs. The demonstration of a bronchostenosis, particularly by bronchography, and especially if the stenosis is irregular and elongated, should lead to the assumption that the pneumonic infiltration is due to carcinoma rather than to a true pneumonia or any real inflammatory process. Such an assumption will usually prove to be correct as illustrated in the first case reported.

DISCUSSION

The findings described above are not by any means completely definitive but must be weighed with the other evidence available. Furthermore, certain cases of carcinoma of the lung will present none of these characteristic findings; many malignancies cannot be identified by roentgen signs. There are, unfortunately, a number of cases which defy all our efforts and must be investigated by exploratory operation. It is obvious that I have pointed out here the positive signs of lung cancer; it is exceedingly difficult to delineate any negative signs. In fact, the absence of carcinoma is almost impossible to establish. But we should not give way to the defeatism which holds that all one sees in the roentgenogram is a shadow from which a definitive diagnosis should not be made. In carcinoma of the lung the roentgen signs are similar in their nature to those observed by the pathologist except that the probability of their correctness is less. That the histopathologic evidence is far more accurate than the roentgenologic no one would dispute. But in the absence of histopathologic evidence we should utilize the roentgen examination to the fullest extent of its potential to make a definitive diagnosis before operation. This is absolutely necessary, not only to institute surgery at a time when it will be effective, but also to avoid indiscriminate surgery, especially in symptomless individuals.

SUMMARY

Roentgen examination may be an effective means of identification, as well as detection, in carcinoma of the lung.

The histopathologic determination of the nature of an abnormal process in the lung may be unavailable or unsuccessful in some cases.

While exploratory thoracotomy is advisable in cases suspected of carcinoma in which positive histopathologic evidence is lacking, it is not always possible to accomplish without more definitive signs than the presence of an abnormal process in the lung.

Intensive roentgen study including comparison with previous films when available, examination in many positions and in different phases of respiration, laminagraphy, and, when necessary, bronchography will produce signs which are highly probable, if not completely definitive for the diagnosis of tumor.

Eleven such findings are described.*

University of Minnesota Hospitals
Minneapolis 14, Minnesota

* For discussion see page 436.

Vol. 74, No. 3

SURGICAL PROCEDURES IN THE DIAGNOSIS AND TREATMENT OF BRONCHOGENIC CARCINOMA*

By EDGAR W. DAVIS, M.D.,[1] SOL KATZ, M.D.,[2] *and* J. WINTHROP PEABODY, Jr., M.D.[3]

WASHINGTON, D. C.

IT IS an overworked truism bordering on the trite that the surgical treatment of carcinoma is most successful when diagnosis is made early. Unfortunately, what is popularly regarded as early diagnosis may actually represent late disease with the unpleasant implication of incurability. Especially is this true when diagnosis depends upon symptoms, for symptoms reflect disturbed function of the involved organ, and all too frequently function is not disturbed, until the malignant process is beyond control.

Carcinoma of the lung, like carcinoma elsewhere, shares this bleak prognosis when diagnosis is based on clinical manifestations. By the time symptoms appear 90 per cent of all pulmonary cancers have extended outside of the lung; theoretical cure is limited, therefore, to 10 per cent of patients in the symptomatic phase.[6] If, on the other hand, surgical intervention antedates the appearance of symptoms, resectability is practically assured, and in 75 per cent of such cases there will be no evidence of lymphatic extension.[6] Encouraging as this may sound, the number of patients seen at this stage are distressingly few, and the over-all picture remains rather discouraging.

The hard, cold facts disclose, for example, that of all patients with bronchogenic carcinoma only 20 to 30 per cent can be resected with any hope of cure and less than 8 per cent will survive five years.[5] Obviously, this can only mean that diagnosis is usually late. This delay is due in part to the patients, many of whom fail to seek medical attention even though they have significant symptoms. Physicians, too, must accept the responsibility for an additional seven months' delay before establishing the diagnosis.[7] Thus, in the average case of carcinoma of the lung, the diagnosis has been made so long after onset that the process is almost sure to have crossed the confines of the lung. Surgery at this stage is indeed not curative. The fault lies not in the limitations of surgery but in the deficiencies of diagnosis, and these are to a certain extent preventable.

Certainly the most valuable single diagnostic aid is the chest roentgenogram, providing that it is taken and assuming that it is properly interpreted. Our ultimate goal, attainable only by still wider utilization of chest roentgen surveys, is the discovery of the early, asymptomatic pulmonary carcinoma. Although peripheral tumors are particularly suited for detection by this means, it is unreasonable to assume that routine chest roentgenograms will ever relieve us of our obligation to recognize potentially serious symptoms. The fact that 95 per cent of patients with cancer of the lung already have symptoms when they consult a physician implies, to us at least, that relatively few patients would take advantage of the opportunity for a chest roentgenogram when bothered by no symptoms whatever. At any rate, our immediate concern is with the need for a more prompt and more thorough investigation of symptoms, not after months of observation, but in their incipiency.

The type and severity of the individual's

* From the Departments of Surgery and Medicine, Georgetown University School of Medicine and the Pulmonary Disease Division of the District of Columbia General Hospital. Presented in a Symposium on Carcinoma of the Lung at the Fifty-fifth Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

[1] Professor of Thoracic Surgery, Georgetown University School of Medicine.

[2] Adjunct Clinical Professor of Medicine, Georgetown University School of Medicine; Chief, Pulmonary Disease Division, District of Columbia General Hospital.

[3] Instructor in Thoracic Surgery, Georgetown University School of Medicine.

Fig. 1. *Diagnosis by Bronchoscopy.* White female, aged forty-four, whom we first saw after four years' treatment for "asthma." Wheeze predominantly right sided. Bronchoscopic findings have been superimposed on normal postero-anterior roentgenogram of the chest. Diagnosis: bronchogenic carcinoma.

symptomatology will vary, of course, with the size and location of the tumor as well as with the degree of bronchial obstruction. The amount of infection distal to the obstruction, the rapidity of tumor growth, the avenue of its extension and the various structures involved, all play an important influencing role. Indeed regrettable is the popular conception of cancer of the lung as a debilitating disease of old men manifesting itself by a constant cough, severe chest pain, frequent hemoptyses, progressive dyspnea, hoarseness and marked wasting. In other words, advanced disease is considered typical, and symptoms, to be impressive, must be severe. Persistent adherence to this concept probably constitutes the chief deterrent to early diagnosis. It is the slight cough, the solitary hemoptysis, the occasional wheeze or barely perceptible dyspnea that must be regarded with suspicion. Chest pain, hoarseness and weight loss are poor prognostic signs and should be viewed with alarm. Pneumonitis secondary to bronchial occlusion occurs often enough to constitute a syndrome demanding careful evaluation. Far too often, because the symptoms are mild or mimic those accompanying an ordinary pneumonia, both the patient and physician are apt to misinterpret their significance, especially when they respond to antibiotic therapy. Recurrent or slowly resolving "pneumonia" is more likely to arouse suspicion, but carries a somewhat more ominous significance due to the additional time lapse.

Depending again upon various factors such as size, location and extent of the malignant process, physical examination may provide confirmatory signs. Inspiratory lag on the involved side in association with a normal percussion note but diminished breath sounds is quite typical. In the presence of infection moist rales and diminished breath sounds are heard. A localized wheeze should always arouse suspicion, as should clubbing of the fingers, particularly when of recent origin. Depending upon the circumstances, evidence of emphysema, atelectasis or pleural fluid may be elicited. In order to obviate needless major surgery, metastatic involvement should be carefully searched for.

So much for the presumptive diagnosis of bronchogenic carcinoma. Once suspected, the exact diagnosis can be established only by certain surgical or semi-surgical techniques. Prompt action is as important here as it is in the initial clinical study of the patient.

BRONCHOSCOPY

Bronchoscopic examination is the most important procedure short of thoracotomy yet available for the definitive diagnosis of pulmonary cancer. By this technique certain centrally situated carcinomas can be visualized directly and a biopsy obtained. Use of the telescopic lens permits better visualization of some of the segmental bronchi, but in our hands has not appreciably improved the incidence of positive diagnoses. When for one reason or another

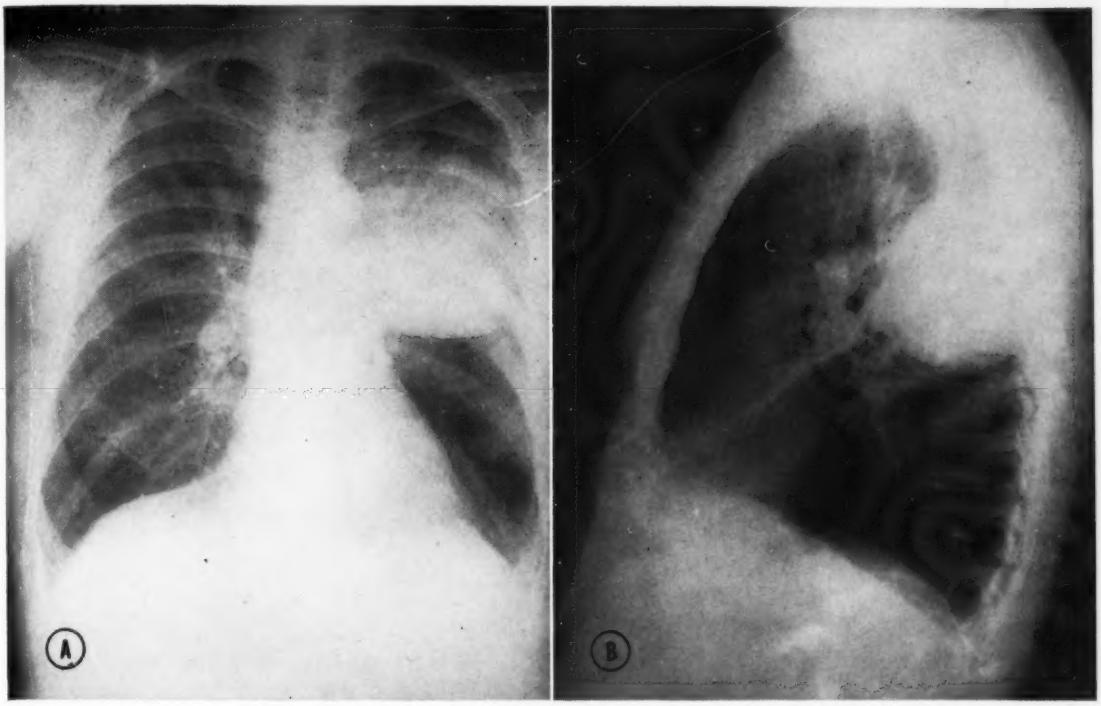

Fig. 2. *Diagnosis by Cytologic Study of Bronchial Washings.* White male, aged fifty-seven, who except for a "cigarette cough" of many months' duration, had no complaints until one week prior to admission when he developed left chest pain on coughing. Chest roentgenograms (*A* and *B*) showed a large mass involving primarily the superior segment of the left lower lobe. At bronchoscopy the superior segmental orifice was completely occluded, but there was no evidence of tumor for biopsy. Bronchial washings, however, contained many malignant cells. Epidermoid carcinoma of the lung was resected one week later.

a biopsy cannot be obtained, bronchial secretions or bronchial washings can be collected from the bronchus or bronchi in question.

Since squamous and small cell bronchogenic carcinomas have a predilection for large bronchi, bronchoscopy is highly successful in demonstrating these lesions, affording direct inspection in about 80 per cent.[4] Adenocarcinomas, on the other hand, affect predominantly peripheral bronchi, which explains why this tumor can be visualized in only 38 per cent of patients.[3]

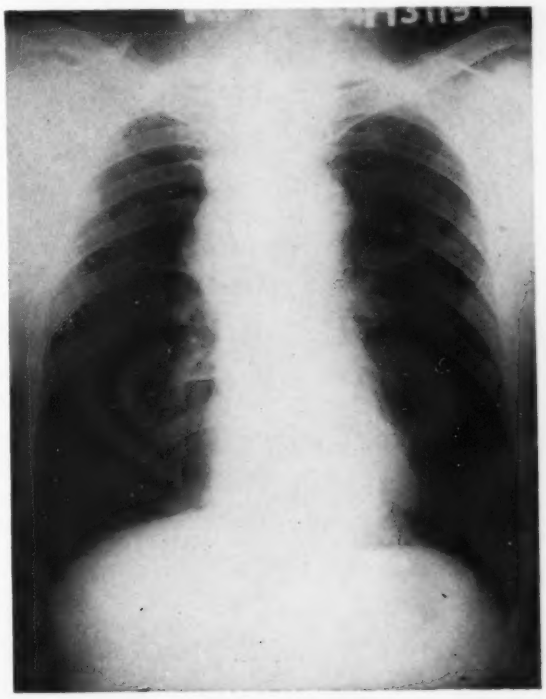

Fig. 3. *Diagnosis by Scalene Lymph Node Biopsy.* White male, aged sixty-one, who complained of a cough of several months' duration. Right paramediastinal mass seen in this roentgenogram of the chest. No cervical lymph nodes were palpable. Bronchoscopy and bronchial washings negative. Biopsy of right scalene lymph nodes showed metastatic anaplastic carcinoma which we consider evidence of unresectability.

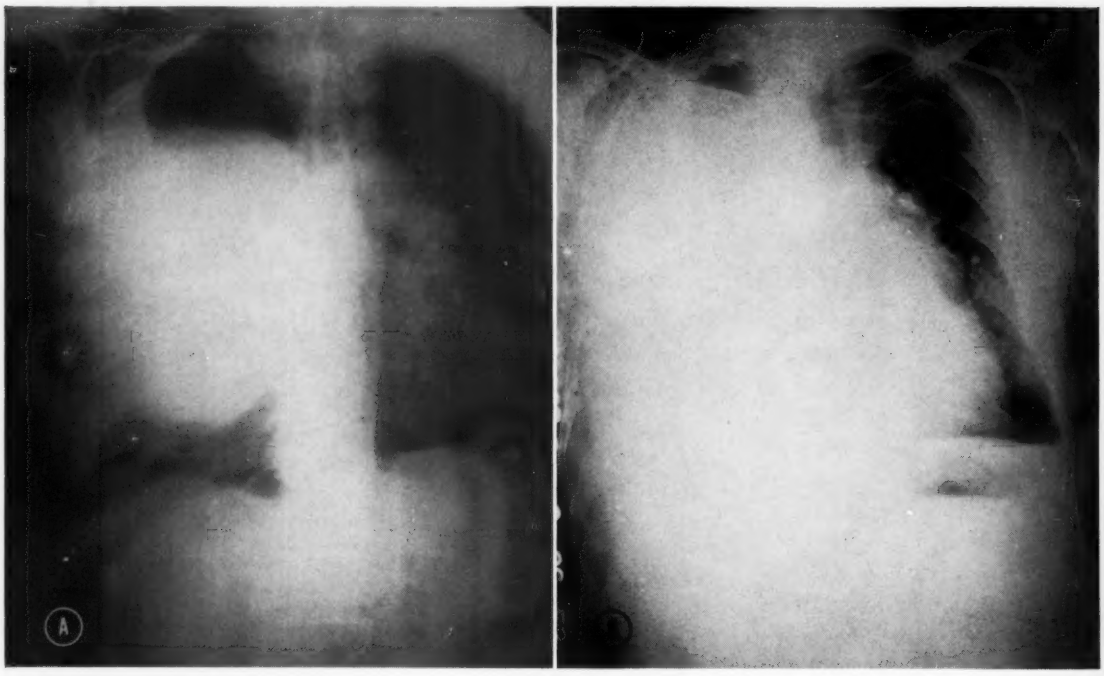

Fig. 4. (*A*) and (*B*) *Diagnosis by Aspiration Biopsy*. White male, aged sixty, with chest complaints for many months. On the basis of an overpenetrated chest roentgenogram (*A*), a presumptive diagnosis of bronchogenic carcinoma was made, but no studies were carried out and surgery was considered inadvisable. Several years later we first saw the patient because of moderate dyspnea. Because of obvious unresectability an aspiration biopsy was accomplished without incident, thus confirming the diagnosis of bronchogenic carcinoma.

Large cell bronchogenic carcinomas occur in large bronchi as frequently as in small ones and are visualized in 50 per cent of cases.[4] Taking all cell types into consideration, direct bronchoscopic inspection of the tumor is possible in about 50 per cent of bronchogenic carcinomas.

The incidence of positive biopsies corresponds to the frequency with which the tumor is seen bronchoscopically. At times, however, although a lesion may be seen, the bronchoscopist may not deem it desirable to perform a biopsy because of the

Fig. 5. *Diagnosis by Cytologic Study of Pleural Fluid.* White female, aged sixty, whose chief complaints had been cough and anterior chest pain for the preceding six months Roentgenogram of the chest revealed a right pleural effusion. Bronchoscopy and bronchial washings were not diagnostic. The aspirated pleural fluid was not bloody but contained malignant cells on cytologic examination. Such cases are not curatively resectable.

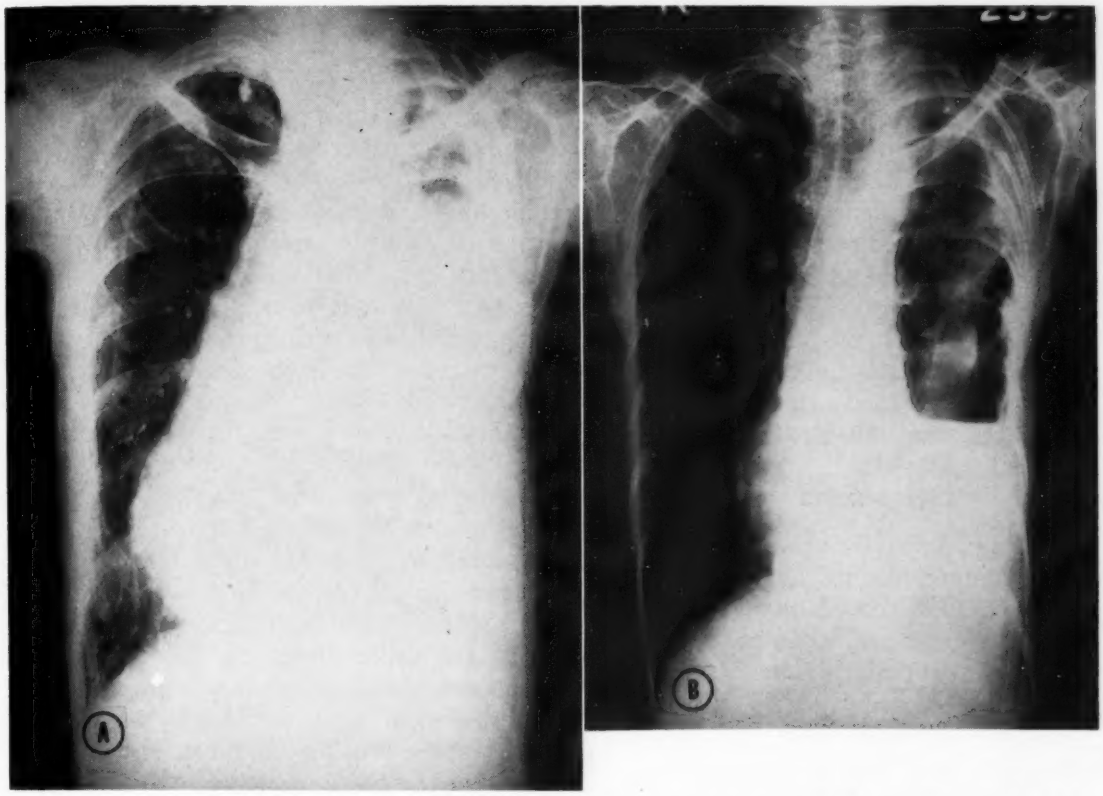

Fig. 6. *Diagnosis by Pleural Biopsy*. White female, aged seventy, with left chest pain of several months' duration. Initial chest roentgenogram (*A*) showed a large pleural effusion. A repeat roengenogram after aspiration of 1,800 cc. of bloody pleural fluid (*B*) disclosed pleural nodularity. Pleural fluid was negative for malignant cells, but a small pleural biopsy established the diagnosis of metastatic adenocarcinoma, which we consider evidence of unresectability.

tumor's vascularity or the possibility of injuring an already damaged bronchus. Furthermore, the portion of tumor that is accessible for biopsy comes from the periphery of the tumor and may be too superficial or too necrotic to be diagnostic. Regardless of how typically malignant the tumor may appear, biopsy should be done whenever possible, for occasionally benign tumors and inflammatory lesions may present a similar appearance.

During the past ten years there has been a striking decrease in the percentage of positive bronchoscopies for bronchogenic carcinoma, due no doubt to earlier diagnosis and more frequent recourse to exploratory thoracotomy for indeterminate pulmonary lesions.

CYTOLOGY

The diagnosis of carcinoma of the lung has been enhanced by the cytologic examination of exfoliated cells in the sputum and bronchial secretions. This method is particularly applicable to tumors which cannot be visualized bronchoscopically. Smears may be taken from the involved bronchus and all secretions collected. When there are no secretions, 5 to 10 cc. of normal saline may be used to irrigate the involved bronchus and the washings examined for malignant cells. Positive findings have been reported in 30 to 90 per cent of cases but our own experience has been a little less satisfactory. Fortunately, false positive diagnoses are rare.

The collection of bronchial secretions

rather than sputum has its advantages. Since they are removed directly from the lesion or close to it, bronchial secretions are more concentrated. Furthermore, the cytologic interpretation of bronchial aspirations is simpler than that of sputum, because there are no superficial squamous cells derived from oral mucosa to confuse interpretation. In all fairness we must admit that the ease and frequency with which sputum can be obtained does much to offset any other deficiency. Ideally, at least three sputum samples as well as bronchial secretions should be examined.

LYMPH NODE BIOPSY

In the presence of a pulmonary lesion of indeterminate nature, biopsy of palpable lymph nodes should always be done. Supraclavicular and axillary lymph nodes are most commonly involved.

So-called scalene node biopsy, which consists of exploration of the retroclavicular space for the purpose of obtaining the deep cervical fat pad with its scalene lymph nodes and any accessible superior mediastinal nodes, is a worthwhile addition to our diagnostic methods. Occasionally we have found it quite useful in settling the question of doubtful resectability. The presence of cancer in these lymph nodes clearly indicates that the operation will be palliative only, and also significantly reduces the likelihood of resectability. Certainly in poor risk patients, scalene lymph node involvement would demonstrate the futility of attempting extirpative surgery.

ASPIRATION BIOPSY

Most thoracic surgeons are of the opinion that needle biopsy should not be done in suspected lung cancer when the lesion is presumed to be operable. Possible complications include infection, hemorrhage, pneumothorax, air embolism and implantation of malignant cells along the needle tract. To our way of thinking these are extremely remote possibilities which are not as important as the problem of false negative results when non-representative tissue is obtained.

Nevertheless, we agree that in several situations, *e.g.*, in patients with apparently inoperable cancer of the lung or in patients in whom surgery is contraindicated but histopathological diagnosis has not been possible, needle aspiration can be of diagnostic value. In all other situations exploratory thoracotomy is preferred not only because it affords a diagnosis but because it constitutes the sole definitive form of therapy.

CYTOLOGY OF PLEURAL FLUID

The presence of pleural fluid in patients with carcinoma of the lung often indicates pleural invasion and incurability. This is especially true if the fluid is blood-tinged. The fluid should be aspirated and the centrifuged sediment embedded in paraffin, sectioned, stained and examined as if it were tissue. Smears can also be studied. Malignant cells are found in about 50 per cent of the cases of pleural effusion due to carcinoma of the lung.[4] Our personal feeling is that patients with massive bloody effusions containing malignant cells are not suitable candidates for surgery, and this applies equally well to the question of palliative resection.

PLEURAL BIOPSY

When examination of pleural fluid fails to disclose the presence of malignant cells, a small intercostal incision can be made and a piece of the parietal pleura removed for histopathologic study. This technique is safe, simple and associated with a low morbidity. Since initiating this procedure we have been very much impressed with the frequency of positive results in selected cases. As with positive pleural fluid studies, pleural biopsies positive for carcinoma comprise, in our opinion, evidence of unresectability.

EXPLORATORY THORACOTOMY

When an accurate diagnosis of carcinoma of the lung cannot be made with the

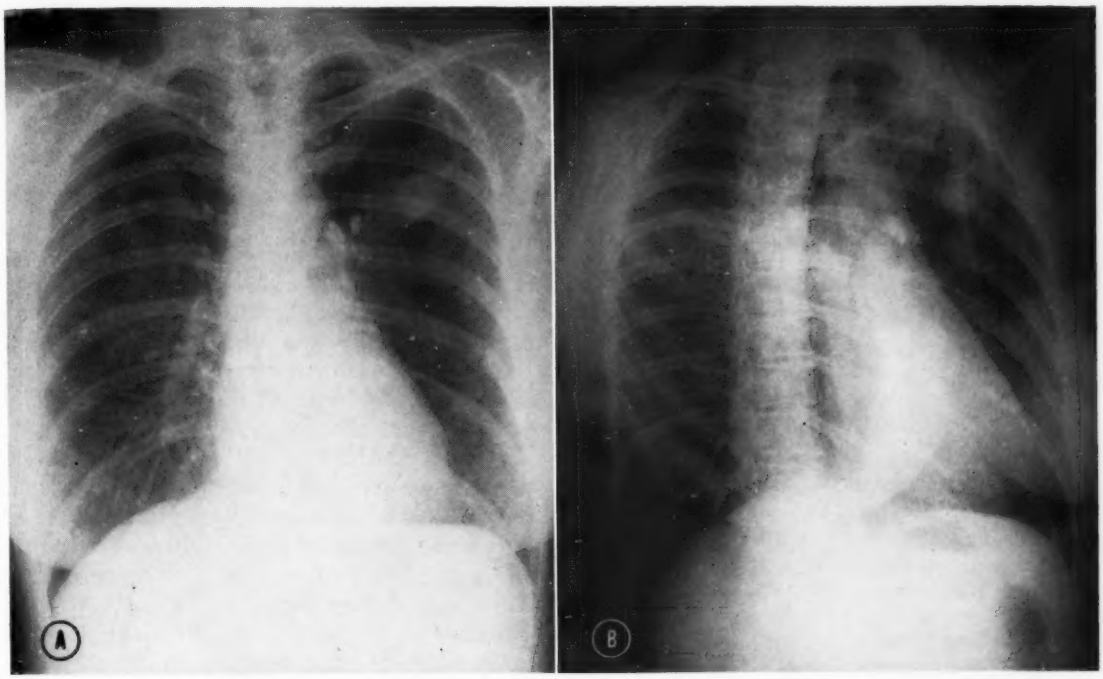

Fig. 7. *Diagnosis by Exploratory Thoracotomy.* White female, aged fifty, who had no complaints whatever. Routine chest roentgenograms (*A* and *B*) revealed a solitary tumor in the left upper lobe. History disclosed the resection of an adenocarcinoma of the sigmoid colon nine years ago. Since that time she had taken a daily dose of mineral oil. On physical examination a mass was discovered in the left breast. Tuberculin and histoplasmin skin tests were both positive. When the breast tumor proved to be benign, a left exploratory thoracotomy was performed with resection of a primary alveolar cell carcinoma of the left upper lobe.

diagnostic procedures outlined, exploratory thoracotomy is the only means of establishing a diagnosis. In approximately 25 per cent of patients with bronchogenic carcinoma involving the larger bronchi, and in practically all patients with reasonably early peripheral tumors, exploratory thoracotomy is the only way in which a diagnosis can be made.[1] When cancer of the lung cannot be excluded, it is exceedingly dangerous to pursue a course of "watchful waiting." For example, the roentgenographic appearance of small peripheral lesions seldom stimulates much concern, yet 50 per cent of all solitary, circumscribed pulmonary lesions will prove to be malignant.[2] Since negative diagnostic studies can be anticipated with this type of lesion, early exploration is imperative. With other types of disease exploration should also be quickly resorted to after appropriate study except when contraindicated by the patient's condition.

SURGICAL TREATMENT

The objective of surgical treatment is the total removal of the tumor and the regionally connected lymphatics. Total pneumonectomy with complete removal of the mediastinal lymph nodes is generally considered to be the only adequate treatment. Some feel that lobectomy is justified when the tumor is well localized within a lobe without involvement of the hilar or mediastinal lymph nodes. Certainly for poor risk patients with impaired pulmonary or cardiac function in whom it is important to conserve functioning lung tissue, lobectomy has ample justification.

When exploratory thoracotomy is used as a diagnostic technique, we strongly advocate a conservative approach restricted to biopsy or a limited resection. In the event that a diagnosis of malignancy is made by frozen section, the remainder of the lobe or lung is removed, depending

upon the extent of involvement and the philosophy of the individual surgeon.

In patients with obviously incurable bronchogenic carcinoma the presence of severe infection distal to bronchial obstruction, severe repeated hemoptyses or uncontrollable pain due to direct chest wall invasion may warrant palliative lobectomy or pneumonectomy.

Edgar W. Davis, M.D.
1150 Connecticut Ave.
Washington 6, D.C.

REFERENCES

1. Clagett, O. T. Exploratory thoracotomy in diagnosis of pulmonary disease. *M. Clin. North America*, July, 1954, 1239.
2. Davis, E. W., Katz, S., and Peabody, J. W., Jr. Surgical implications of solitary tumors of the lung. *Am. J. Surg.*, 1955, *89*, 402.
3. McDonald, J. R., McBurney, R. P., Carlisle, J. C., and Patton, M. M. Significance of cell types in bronchogenic carcinoma. *J. Thoracic Surg.*, 1951, *22*, 62.
4. Moersch, J. H., McDonald, J. R., and Holman, C. B. The diagnosis of bronchogenic carcinoma. *M. Clin. North America*, July, 1954, 1109.
5. Ochsner, A., DeCamp, P. T., DeBakey, M. E., and Ray, C. J. Bronchogenic carcinoma; its frequency, diagnosis, and early treatment. *J.A.M.A.*, 1952, *148*, 691–697.
6. Overholt, R. H. Cancer detected in surveys. *Am. Rev. Tuberc.*, 1950, *62*, 491–500.
7. Overholt, R. H., and Schmidt, I. C. Silent phase of cancer of lung. *J.A.M.A.*, 1949, *141*, 817–821.

DISCUSSION OF SYMPOSIUM ON CARCINOMA OF THE LUNG

(Papers by Averill A. Liebow, L. Henry Garland, Leo G. Rigler, and Edgar W. Davis, Sol Katz, and J. Winthrop Peabody, Jr.)

Dr. Aubrey O. Hampton, Washington, D.C. As has already been stated by Dr. Davis, these papers are extremely meaty and complete. As one member of this Society, I do not believe that smoking is such a foul habit. If you look at the graphs that were presented here you will note that they were parallel with the bottom line, whatever that line is, from 1800 to 1920. Then suddenly the graphs took an upward swing. I believe that was because of progress in radiology.

I remember very well the time when we didn't know how to make a diagnosis of collapse of a lobe. We called it postoperative pneumonia, when it was postoperative atelectasis.

Dr. Holmes and Dr. Churchill, I believe, described the first case of collapse of a lobe and roentgen findings, and that was in the early 1920s. Such progress as has been presented here by Drs. Garland and Rigler in radiology, and Dr. Davis in surgery, I believe, accounts for a great deal of the increase in carcinoma of the lung. Of course survey films of the chest have, too. There are all kinds of reasons why our ideas are not absolutely correct when we say there has been an increase. I know I am in the minority, and I don't mind that at all.

I would like to know why this nice, discrete disease we had called adenomatosis of the lung, which did not metastasize, now metastasizes. There was a time when we had a very nice disease that radiologists could diagnose, and the patient did not have metastasis at postmortem. They just died of asphyxia.

I followed one good friend of mine to his death, and he had no metastases. I understand now that they are all mixed in with broncheolar carcinomas.

I would like to know why Dr. Davis reports an incidence of 50 per cent of carcinoma in so-called coin lesions which are not calcified, and Dr. Garland's soars all the way from 4 to 40 per cent. He is quoting other people. I don't know what his own figures are.

If these rounded nodules cannot be identified except by Rigler's umbilicated notch (and I haven't seen one yet), then what are we going to do about it? Are we going to follow them until, as Garland says, it is too late? He says it is too late when we find them. Certainly if we follow them it will be too late.

I am finding myself in a very embarrassing situation. I am going to do everything Rigler suggests, mind you, and I am doing that; but I still don't end up with the answer frequently.

Dr. Brian Blades, Washington, D. C. The participants in the Symposium have covered the subject so completely there is little for a discussant to add.

I should like to mention one factor in particular which was not emphasized, namely the indolence of growth in some bronchogenic carcinomas. The coin lesion or any other type of opacity may be seen in a roentgenogram of

the chest and reported by the radiologist and later the family doctor may obtain follow-up films and there is no change in size of the opacity. The roentgen report will likely say, "No change." This may give the physician a false sense of security. Months or perhaps years may go by without change and when the change does occur the lesion may be inoperable.

I doubt that any radiologist would care to make a complete and final diagnosis upon looking at a roentgenogram, but I wonder if it would not be a good policy to suggest the possibility of tumor in these cases. The problem of the patient with a pneumonia-like shadow which responds to antibiotic therapy but really has a lung cancer has been mentioned. Dr. Davis has mentioned a figure of 25 per cent or so of pneumonia-like shadows in his series. That corresponds very closely to our figures and, even more important, is that in 24 per cent of our cases positive action was delayed because there was a temporary improvement on antibiotic therapy.

It is difficult to discuss this subject without mentioning smoking. It seems to me that this problem is not completely settled. I have wondered many times if the factors which make the patient a chain smoker, the endocrine drive or whatever this may be do not have more to do with the production of cancer than the smoking itself.

Dr. Ross Golden, Los Angeles, California. You have heard this morning some authoritative discussions of a serious problem which is confronting the medical profession at the present time. The question was raised as to whether carcinoma of the lung is actually increasing in incidence. I remember hearing this discussed at a meeting at the New York Academy of Medicine some fifteen years ago. One speaker pointed out that, in the autopsy records of Bellevue Hospital, the occurrence of cancer of the lung was rare during the first decade of this century. In the fourth decade it was much higher. He felt that this could be taken as reliable evidence that the incidence of cancer of the lung was actually increasing without involving the question of better diagnosis. If cancer of the lung continues to increase, or at least if we continue to detect increasing numbers of carcinomas of the lung as we are doing now, this disease is going to develop into a major disaster in another quarter of a century because it will kill so many middle-aged, important men.

A great deal has been said about early diagnosis. Unfortunately, we cannot be sure that a carcinoma is early from the biological standpoint, because we cannot tell whether or not it has metastasized. We are therefore left with only one criterion, and that is the size of the cancer; and our objective is to find these cases while the cancers are small. Until a reliable serological test for malignant disease has been developed, the only practical method by which this can be done is by roentgen examination.

Dr. Rigler discussed certain roentgenologic methods of examination of the chest. He mentioned laminagraphy several times. This valuable technique is not used nearly as much here in the United States as it is in Europe. We should use it much more than we do, but unfortunately we have to go to Europe to get good equipment. If I am not mistaken, only one apparatus for transverse laminagraphy is in use in this country at the present time.

Although I personally have not had anything to do with mass surveys, from ordinary clinical experience I am now convinced that the so-called lordotic projection is a valuable addition to the routine posteroanterior film for the detection of small lesions in the upper half of the lung. I wonder whether it would not be a good idea to run a series of survey cases in which the lordotic position is used along with the routine posteroanterior projection to determine whether or not it will reduce the number of failures to detect small lesions.

Another technique which is impossible in survey work, as Dr. Garland said, is the use of supervoltage (1 to 3 million volts) roentgen rays. From the cases studied by Dr. Pendergrass and his associates, it seems that this will at some time prove to be an addition to the clinical study of cases with lesions in the lung.

On September 10 and 11, 1954, one week ago last Friday and Saturday, the American Cancer Society held a conference on cancer of the lung, which was attended by approximately thirty-five people interested in various aspects of this problem. The deliberations, well organized by Dr. Charles Cameron, were divided into two sections, one dealing with research on the cause of lung cancer, and the other with clinical problems of detection and treatment. The research group presented some very interesting investigations, both statistical

and experimental, on the association of cancer of the lung with the smoking of cigarettes. This was sufficiently impressive to cause the conference group to recommend that the evidence on this question be publicized by the American Cancer Society and the U. S. Public Health Service, to the end that the public may be warned of this danger. This recommendation was made in spite of the fact that several of those present were actually smoking cigarettes while they voted "yes." There was no demonstrable relationship between cancer of the lung and smoking of pipes and cigars. A possible relationship to other inhalants, such as fine particles of asphalt and automobile exhaust fumes, was discussed, but it seemed to be the opinion of the investigators that evidence incriminating these substances is not definite at the present time.

The American Cancer Society has prepared a bibliography on the causes of lung cancer. It includes fifty-two references on the possible relation of this disease to tobacco smoking. Only two of these papers were published before 1950. I am sure the American Cancer Society would be glad to make this bibliography available to any interested physician.

The clinical group at this conference was of the official opinion that as "early" a diagnosis as is possible is of importance in the control of cancer, in spite of some of the pessimistic opinions we have heard, and that retrospective studies should be done whenever possible, such as Dr. Rigler has illustrated. They also recommended that pilot studies of x-ray screening programs be done in selected areas to get evidence on the long-range value of this procedure as a life-saving measure. Reduction in mortality, as has been pointed out this morning, is quite a different problem from that of detecting lesions by survey methods.

Dr. Rigler has emphasized the importance of comparing present with previous films. Unfortunately, most radiologists in private offices, and even in many hospitals, cannot store films very long. What can be done to insure the availability of previous films? Can the average patient be entrusted with his chest film if properly instructed as to its value and can he be told to take it with him the next time he goes to a doctor? The answer to this problem remains to be found.

Other diagnostic procedures, such as bronchoscopy, laminagraphy, etc., can be done only after the presence of disease in the lung has been demonstrated either by a survey film or by a roentgen study of a patient with symptoms. The value of these procedures is unquestionable and certainly they all should be done if a patient has a shadow in the lung. But if these refined procedures fail to give unequivocal evidence of cancer and if the shadow in the lung has even a 50–50 chance of being a cancer, I think the patient deserves exploration by a qualified thoracic surgeon, unless he has serious heart disease or some other physical handicap. Under normal conditions, in the hands of surgeons like Dr. Davis and others, who are becoming increasingly available, the risk of exploration of the lung is very slight. If a shadow which could be a cancer, turns out to be a tuberculoma, I think that patient can consider himself very lucky.

DR. GARLAND (closing). To answer Dr. Hampton's question, in my own experience in a survey made in the San Francisco area, about one-fourth of the circumscribed shadows turned out to be bronchogenic carcinomas or malignant tumors. We have had quite a few coccidiomas in our part of the country, and the rest were miscellaneous granulomas, hamartomas, and so on. The cases we have seen cured had an average diameter of about 4 cm. In other words, the lesion was *not* small but was still localized (as are a few bulky carcinomas of the stomach or breast). However, they are exceptional; our local surgical cure rate is still under 5 per cent.

A "mature man" has been defined as one willing to face all of the available facts, however disagreeable they may be. When I was first faced with the disagreeable fact of the large element of error in chest roentgenology, I didn't believe it. I was rudely awakened. Photofluorograms are not the precise instruments which many non-radiologists appear to believe. *They are the best available survey weapon today, but they are very crude.*

Some persons would like to have official health agencies undertake a permanent program of free chest x-rays. Assuming for a moment that this were possible, it was asked in a recent Lung Cancer Conference, "If the original x-rays are free and are to be followed by prompt action, should not the follow-up services on tumor suspects also be free up to and including thoracotomy?" Some of the surgeons present demurred!

The medical profession in general still be-

lieves that the *diagnosis*, as well as the *treatment* of cancer, is still a function of medical practice in this country; and it is my own impression, from a long experience with the Veterans Administration and the U. S. Public Health Service, that if this screening procedure were conducted by governmental or national agencies it might easily do more harm than good.

The second fallacy which I tried to emphasize this morning, and which is expounded endlessly by some of my good surgical friends, is the assumption that early cancer is the same as localized cancer. To most persons "early" connotes "asymptomatic" or "small," but in actual fact roentgenologically small or early lung cancer is frequently no longer localized or curable. The silent lung cancer only 2 cm. in diameter often has metastasized beyond the realm of resection.

Well, we have these facts. Now it is our job to go ahead and try to prevent this disease, or to develop some other better means of detection.

May I thank the discussants for their remarks.

Dr. Rigler (closing). There are three points I would like to take issue with Dr. Garland on, and first is the question of pessimism with regard to surveys. If one critically examines what the results of the surveys are, it is perfectly evident that we have failed miserably—but the failure, I think, lies with us and not with the method, and I believe we ought to appreciate that. I think we have been overly conservative in the interpretation of photofluorograms. I don't think it is in the photofluorogram itself but in our own interpretation of it.

Carcinoma of the lung is indolent in a very high percentage of cases, not in the exceptional case, as shown by the figures we have accumulated in 80 cases—a small number, it is true, but still a group followed very closely.

These were accumulated through a retrospective study, in which we sent out for films made previously, and we found that in this group of cases the average duration between the first roentgen evidence of disease, as seen in a film, which eventually proved to be carcinoma in the same area, and the time of the death of the patient, was something like twenty-five months.

Bear in mind that we have taken into consideration every film made three months or more before we saw the patient. These films were fortuitously made, and in many of them the lesion was already very large; if a film had been made earlier the lesion would have been much smaller, and we don't know how many years had gone on up to that point.

If you take that into consideration you will realize that those twenty-five months are an absolute minimum, and that it probably is much longer. This is an average, and I think it indicates that this is not an extremely malignant disease, on the whole, which rapidly goes to its final conclusion.

Of course there are within the group malignant lesions, and this idea of biological predeterminism and the pessimism which all of us feel because of that must be considered. But I still think, in view of the experience we have just had with carcinoma of the stomach that much can be done if the lesion is treated when it is small. We have reviewed 75 patients who were seen at a stage when they were either asymptomatic or in which by reason of the findings in the films we could be certain the lesion in the stomach was small. In that group of cases we have determined the frequency of involvement of lymph nodes, which is by far the most crucial point insofar as the salvage from carcinoma of the stomach is concerned—in that group the rate of involvement of lymph nodes was one-third that of a symptomatic group of carcinomas of the stomach.

In other words, three times as many patients had lymph nodes who had symptoms, as those who were found by routine roentgen examination. I submit that this means that while there undoubtedly is a biological predeterminism, it is not as potent as it might be.

We might look at the figures in carcinoma of the lung for just one moment. The general figures which are given in symptomatic patients with carcinoma of the lung are that the cure rate at the end of three years is something in the range of 6 per cent. This is about as good as is generally accomplished. The figure Dr. Overholt presented in a small series of asymptomatic, survey-discovered cases is a 30 per cent five year curve.

I must say that those additional 24 per cent really amount to a great deal. It isn't just that 30 per cent isn't a good figure, but the difference between 6 and 30 is a very substantial figure in the life of any individual, Even the difference between 6 per cent and 11 per cent, the figure

Dr. Garland used, is a very substantial one and certainly not to be dismissed lightly.

DR. DAVIS (closing). In answer to Dr. Hampton's query about the incidence of malignancy among our solitary pulmonary tumors, let me first explain the figures to which he refers and then mention a corroborative study that we have subsequently carried out. The point in question is the isolated, innocent-looking, circumscribed nodule which from its roentgenographic appearance once used to be referred to rather glibly as a "tuberculoma." As early as 1947 we presented a paper stressing the malignant potentialities of this lesion. By 1949 we were able to report 67 consecutive patients with surgically excised solitary nodules of the lung, 37 (55 per cent) of which proved malignant.* Inasmuch as this figure is often cited as the highest in the literature, and lest it represent a sample falsely weighted with carcinomas, Dr. Peabody, my associate, felt that an analysis of the next 67 similar cases might provide an interesting comparison. This study, published elsewhere,† disclosed a remarkably close correlation between the two series, since there were 34 malignant nodules among the second group, an incidence also slightly in excess of 50 per cent.

Obviously then, any solitary tumor of the lung deserves excision except under extenuating circumstances. Chief among these is the presence of calcium which can be considered a dependable though not infallible guide to benignity. Of course, an increase in size, the development of cavitation or associated symptoms may negate the reliability of this sign.

In closing, I have only three comments. First, like Dr. Rigler I consider the term "coin lesion" quite inappropriate. Being accustomed, as I feel we all should be, to the routine use of lateral and oblique views, it is a little annoying to have only the posteroanterior appearance taken into consideration. To call a nodule a coin, though a mere figure of speech, is a misleading misnomer that might best be discarded. Secondly, I cannot let Dr. Garland go unchallenged in his intimation that the surgical results are not improved by earlier diagnosis. Too often, it is true, the small carcinoma has already metastasized. To me, however, the more important concern is for those which have not yet metastasized but would do so if left alone for another month or two. Granted that an absence of symptoms is no guarantee of curability, it does practically assure the resectability of a lesion and certainly improves one's chance for survival. There are advantages therefore to earlier diagnosis. Finally, if compelled to take a stand on the purported carcinogenic effect of cigarettes, I must admit that to date I retain both the smoking habit and the pleasure that goes with it.

I would like to compliment the various speakers upon what has been a most thorough yet unusually lucid presentation of all phases of the problem of carcinoma of the lung. At the same time let me express my appreciation for the opportunity to participate in this splendid symposium.

* Davis, E. W., and Klepser, R. F. Significance of solitary intrapulmonary tumors. *Surg. Clin. North America*, 1950, *30*, 1707.

† Davis, E. W., Katz, S., and Peabody, J. W., Jr. Surgical implications of solitary tumors of the lung. *Am. J. Surg.*, 1955, *89*, 402.

Vol. 74, No. 3

PULMONARY ARTERIOVENOUS FISTULAS

By WALTER J. STORK, M.D.
HOUSTON, TEXAS

DURING recent years methodical and meticulous examination of the lungs has added much to our knowledge of certain vascular abnormalities. These may be symptomless and thus not detected except by accidental finding during a physical or roentgenological examination. From recent experience it has become increasingly evident that many conditions previously regarded as tumors actually have manifested characteristics of vascular malformations.

Churton[3] as early as 1897, and Wilkens[14] in 1917, recognized pathological communication between arteries and veins of the lungs at autopsy. The first reported clinically diagnosed case has been generally credited to Rodes[13] in 1938. Forty-four cases were listed by Yater, Finnegan and Giffin[15] in their review of both foreign and domestic literature in 1949. Biampalmo[2] reviewed 57 cases only one year later. More frequent exploration of the thoracic cage and imporved surgical technique in recent years have played important roles in this sudden impetus to the discovery of these vascular malformations. In addition, some credit must be given to mass chest photofluorography conducted in many areas.

CLINICAL MANIFESTATIONS

Reading[11] in 1932, described the cardinal triad of clubbing of the fingers and toes, polycythemia and cyanosis in a patient who died of a brain abscess and at postmortem proved to have a pulmonary hemangioma. Both congenital and acquired arteriovenous fistulas are reported in the literature. The acquired fistulas usually are traumatic in origin. The congenital type nearly all belong to the heredofamilial angiomatoses. The disease occurs in both sexes, predominating in persons twenty to thirty years of age. The symptoms may have their origin in childhood or there may be no symptoms throughout life.[12] The clinical characteristics and symptoms are dependent upon the fragility of the involved vessels and the site of the pathological communication.

The chief symptoms, aside from clubbing of the fingers and toes, are apparently due to chronic anoxemia produced by incomplete aeration of the blood in the lungs. This stimulates the erythropoietic system resulting in polycythemia. Secondary symptoms to the polycythemia may occur in variable degree, such as dyspnea, numbness, fainting, diplopia, thick speech, hemoptysis, epistaxis, emesis, and chest pain. According to observations made by Madden and Zion[7] approximately 30 per cent of the blood must be shunted before cyanosis becomes manifest in otherwise normal individuals. The degree of polycythemia has been observed to be in direct proportion to the size of the fistula. There is usually no significant change in the size of the heart. The lack of cardiac enlargement in these cases is attributed by Moyer and Ackerman[10] to the fact that arteriovenous fistula is confined to the pulmonary circuit where a low pressure exists. However, Baer and his associates[1] report a case which showed decrease in the heart size after removal of the pulmonary fistula. A continuous murmur over the lesion is audible in approximately 50 per cent of the cases. The dull zone on percussion is a helpful clinical aid in the diagnosis. In spite of the characteristic clinical syndrome of this disease it is occasionally mistaken for similar congenital cardiopathies and polycythemia vera.

Complications of pulmonary arteriovenous fistulas are often dramatic. Fatal hemoptysis, as in Case 1 herein reported, may ensue and, as in 4 other reported cases, result in death. Cerebral complications are frequently annoying. A greater per cent of these patients complain of headache, convulsive seizures, dizziness, and hemiplegia. Moyer and Ackerman[10]

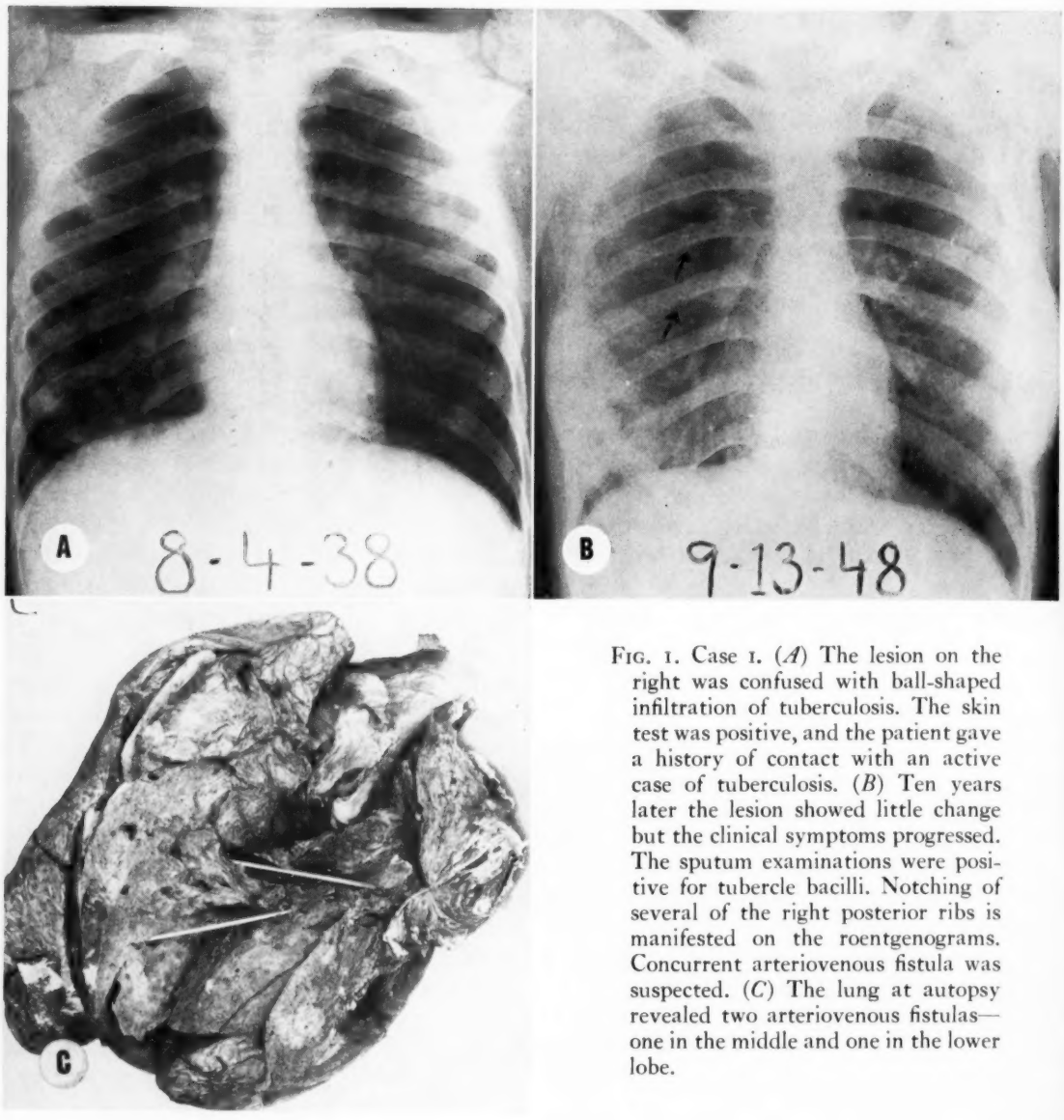

FIG. 1. Case 1. (*A*) The lesion on the right was confused with ball-shaped infiltration of tuberculosis. The skin test was positive, and the patient gave a history of contact with an active case of tuberculosis. (*B*) Ten years later the lesion showed little change but the clinical symptoms progressed. The sputum examinations were positive for tubercle bacilli. Notching of several of the right posterior ribs is manifested on the roentgenograms. Concurrent arteriovenous fistula was suspected. (*C*) The lung at autopsy revealed two arteriovenous fistulas—one in the middle and one in the lower lobe.

stressed the occurrence of postoperative hemorrhage and phlebothrombosis. They warned against possible vascular thrombosis in the untreated case.

Anatomically the pulmonary lesion consists of angiomatous malformations with arteriovenous anastomoses. As a result of these, a shunt develops in the lesser circulation which bypasses the aveolar system. The pathological studies of the specimen usually reveal a large thin-walled vascular sac which consists of a distended afferent artery and distended efferent veins. Between the arterial and the venous systems there is either a distinct communication through one or more larger vessel trunks, or a tangle of more or less distended vessels, instead of capillaries. Degenerative changes arising in the walls are seen and in some places appear actually to present ruptures. From this it might be concluded that other pathological communication channels could arise and thus increase the circulatory disturbance. If a rupture in the vessel

has occurred, hemorrhage in the surrounding tissue is obvious. Moyer and Ackerman reported multiple fistulas as well as other anomalies in the lung of one of their cases.

The most common sites for these fistulas and other vascular abnormalities are between the lobules, along the intralobular and bronchovascular bundle, and below the interlobar and mediastinal pleura. The rich and complex vascular organization of the lung during fetal life apparently provides a favorable basis for arteriovenous anastomoses.

ROENTGENOLOGICAL MANIFESTATIONS

Pulmonary arteriovenous fistulas have been recorded and described in sufficient numbers to establish certain roentgenological diagnostic criteria. One or more rounded, cylindrical, nodular or slightly lobulated opacities are seen on either or both sides, more commonly in the lower lobes. Nearly always these vascular masses are connected with the hilar structures by at least two cord-like often tortuous and irregular densities (Fig. 3). These represent the pulmonary artery and the dilated pulmonary vein, both of which open into the tumor-like vascular sac. There may be more than the two abnormal vessels so frequently seen. Invariably there will be noted evidence of pulsation and increased density of the hilar structures on the involved side. Usually the heart is normal in size.

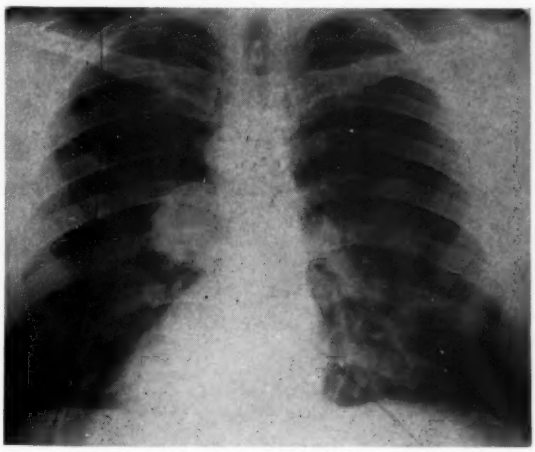

Fig. 2. Case II. The clinical impression did not suggest the diagnosis of arteriovenous fistula, yet the roentgenological appearance is that of a large vascular tumor. Angiocardiogram outlines the aneurysmal wall and the pulmonary artery. This is a pulmonary artery aneurysm and not a fistula.

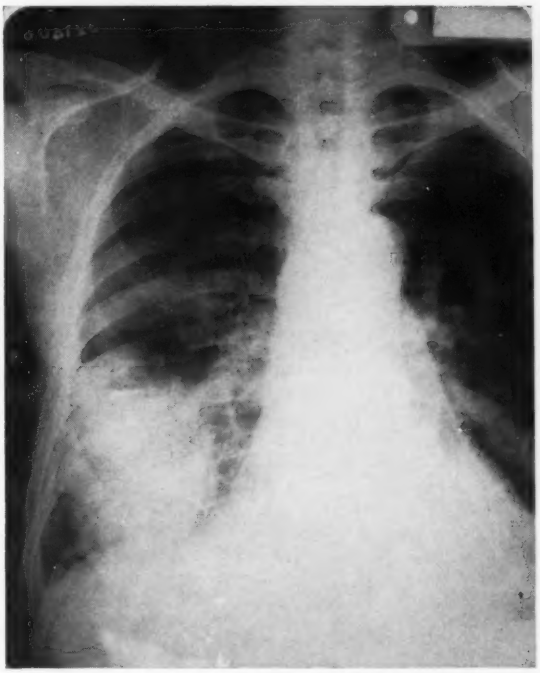

Fig. 3. Case III. The large opacity shown on the right with at least two band-like communicating shadows leading to the dense hilar structures proved to be a pulmonary arteriovenous fistula at operation. The diagnosis is made on the roentgenogram alone.

Roentgenoscopy usually reveals pulsation of the tumor and must always be included to complete the examination. It invariably will show pulsation and increased density of the hilar structures on the involved side. In the author's experience this pulsation may be so great that the expansile motion of the tumor is not easily differentiated from transmitted pulsation. The tumor may not pulsate because of a clot. In the peripheral lesion it may not be visible without the use of roentgen kymography. The tumor may show variation in size, depending upon

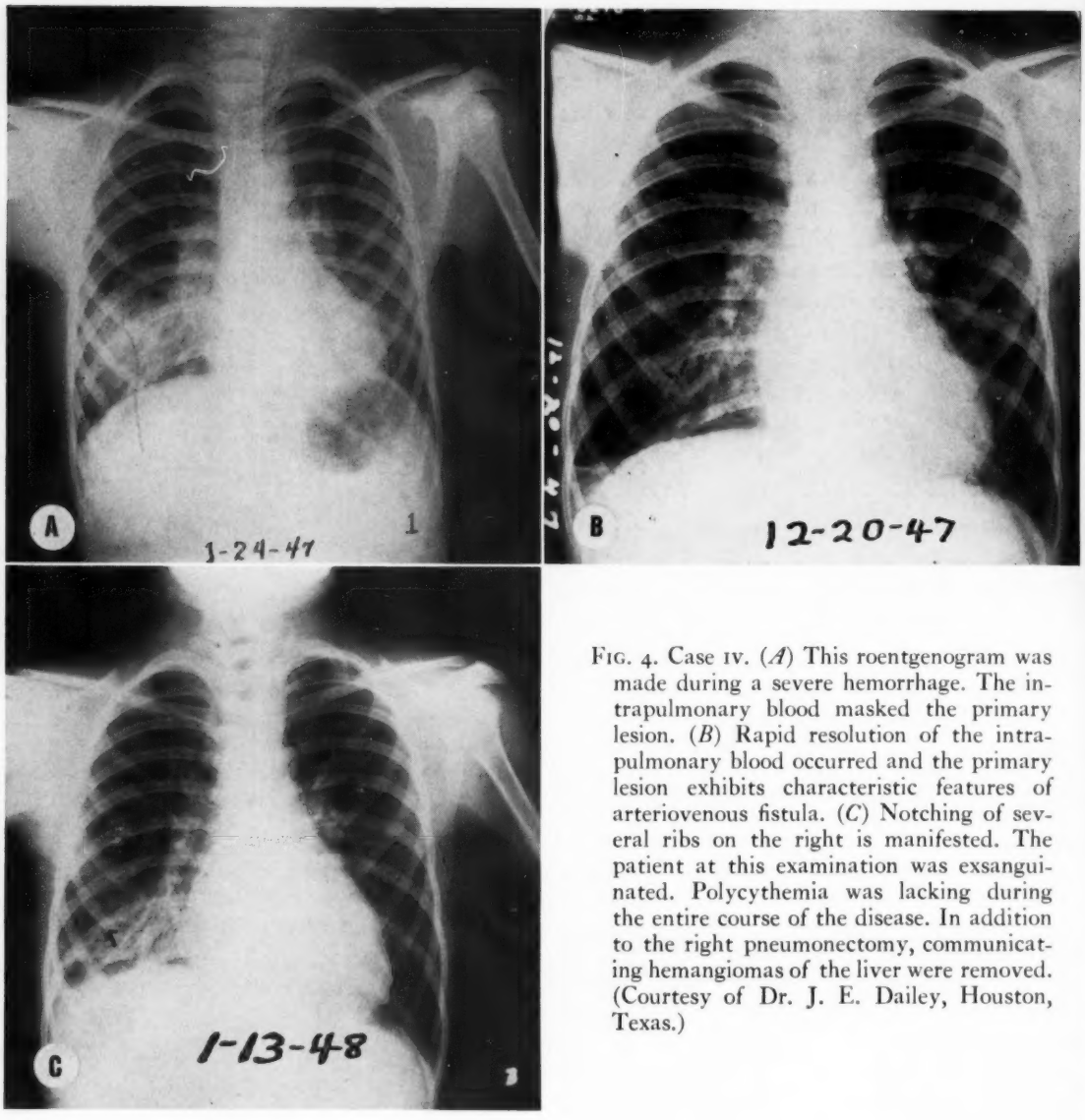

FIG. 4. Case IV. (*A*) This roentgenogram was made during a severe hemorrhage. The intrapulmonary blood masked the primary lesion. (*B*) Rapid resolution of the intrapulmonary blood occurred and the primary lesion exhibits characteristic features of arteriovenous fistula. (*C*) Notching of several ribs on the right is manifested. The patient at this examination was exsanguinated. Polycythemia was lacking during the entire course of the disease. In addition to the right pneumonectomy, communicating hemangiomas of the liver were removed. (Courtesy of Dr. J. E. Dailey, Houston, Texas.)

change of the intrathoracic pressure. The Valsalva maneuver (deep inspiration, followed by forced expiration against the closed glottis) causes the mass to become smaller. With the Müller maneuver (deep expiration, followed by forced inspiration against the closed glottis) the mass increases in size. As Moyer and Ackerman[10] have pointed out, this change in size may be minimal and thus hard to detect. The maneuvers should always be followed by roentgenograms.

Stereoroentgenograms usually are necessary. The communicating vessels, however, lie in different planes. Several projections must be made to demonstrate the relations of the abnormal vessels and the tumor, produced by this lesion. Serial laminagrams made in the lateral projections are most helpful (Fig. 9). Following the formation layer after layer in both the anterior and right angle projections in order to ascertain whether the opacity merges into a vessel and, in this case, into which vessel, aids immeasurably. After operation such an anatomical follow-up is also advisable.

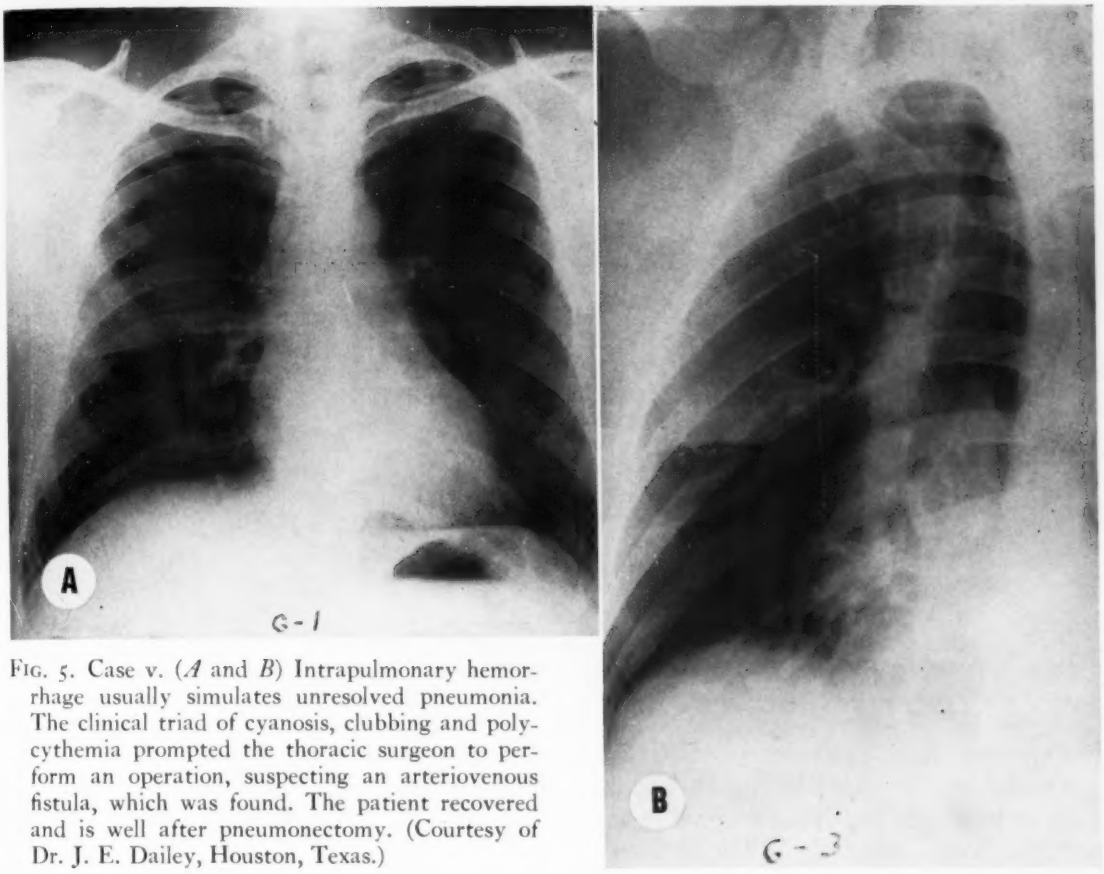

Fig. 5. Case v. (*A* and *B*) Intrapulmonary hemorrhage usually simulates unresolved pneumonia. The clinical triad of cyanosis, clubbing and polycythemia prompted the thoracic surgeon to perform an operation, suspecting an arteriovenous fistula, which was found. The patient recovered and is well after pneumonectomy. (Courtesy of Dr. J. E. Dailey, Houston, Texas.)

Angiocardiograms demonstrate clearly the vascular character of the tumor and its connections with the pulmonary vessels. It also may demonstrate more fistulas than suspected. Even the opposite side may be involved (Fig. 8). It then would seem of utmost importance to do routinely an angiocardiogram on all cases before an operation is attempted, to determine the location and number of lesions. The lesions shown by routine roentgenograms are usually the largest fistulas, but not necessarily the only ones (Fig. 8). Extreme basal location of these lesions may be obscured by the density of the diaphragm or the cardiac silhouette. Overexposed roentgenograms and oblique views must often be made to illustrate these clearly (Fig. 6). Arterial catheterization for determination of changes in the components of the blood is a valuable procedure but is not always necessary to complete the diagnosis. Moyer and Ackerman[10] suggest that angiocardiography be done after catheterization. They caution against the danger of possible venous thrombosis because of the increased blood viscosity in marked polycythemia. Some medical centers resort to anticoagulants during the procedure of angiocardiography to prevent phlebothrombosis.

The roentgenological diagnosis may not be easy. Small cysts, mesotheliomas, adenomas, bronchogenic carcinomas, metastatic lesions, tuberculomas, hernia of the diaphragm, pulmonary infarct, unresolved pneumonia, bronchiectasis, pulmonary artery aneurysm, ball-shaped infiltration of tuberculosis, and even hamartomas are all lesions which must be differentiated. The problem often is resolved if the cardinal roentgenological criteria are recognized:

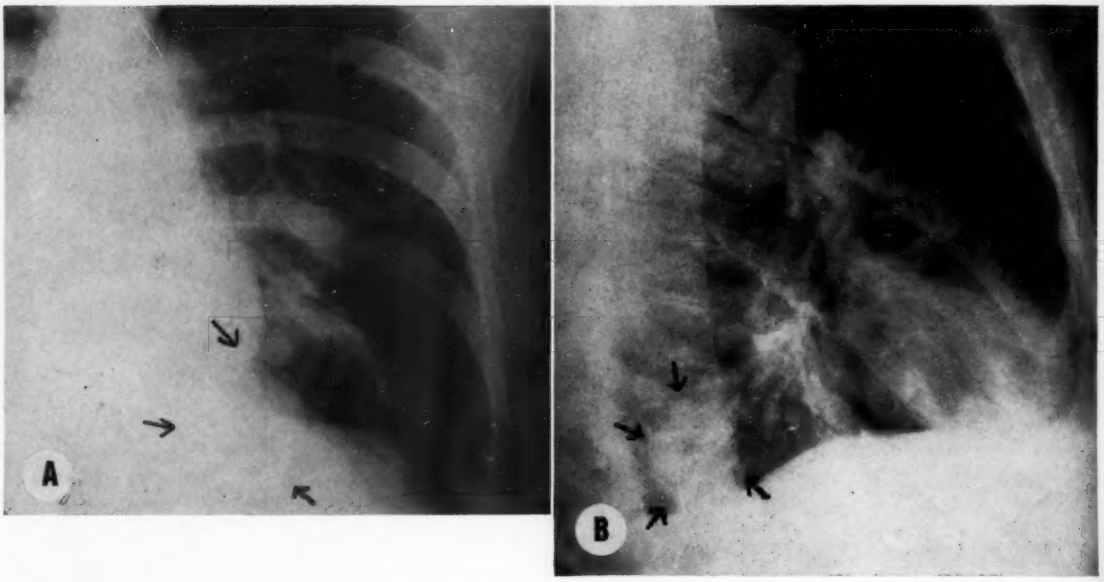

Fig. 6. Case VI. (*A* and *B*) This patient had the typical clinical syndrome and indicative roentgenological findings of arteriovenous fistulas of the right lower lobe, which were substantiated at operation. The lesions are partly overshadowed by the cardiac silhouette and the diaphragm in the posteroanterior projection. They are better visualized in the overpenetrated roentgenogram.

one or more opacities on either or both sides, more commonly found in the lower lobes, with at least two band-like communicating densities connecting the hilar structures, and the invariable pulsation and increased density of the hilar structures on the involved side. This alone should indicate the diagnosis. These features may not be discernible in every case or they may be simulated by other lesions. Aneurysm of the branches of the pulmonary artery without a shunt perhaps offers the greatest problem (Fig. 2). Lindgren[6] believes that the distinguishing point is the vessels which are more distended in the fistula. He also stresses the importance of demonstrating the vascular origin of the opacity to distinguish pulmonary artery aneurysm from other lesions. An aneurysm of the pulmonary artery without a shunt does not cause cyanosis, which is usually present in arteriovenous fistula. Errors in diagnosis have arisen because aneurysms may not show pulsation as a result of clotting, which tumors may show transmitted pulsations; these difficulties are readily resolved by angiocardiography.

Tuberculomas are usually not so large as the arteriovenous fistulas. They may be arranged in chain, thus giving the appearance of a lobulated mass from which frequent bronchovascular markings are seen leading to the hilar structures (Fig. 10). These lesions frequently show signs of increased density or central calcification if they do not excavate (Fig. 11). In the event they do excavate, the dissemination usually progressively advances to be readily detected on the roentgenogram. Tuberculomas always lack the hilar pulsation on the affected side. Ball-shaped infiltration of tuberculosis may offer a problem, especially in the presence of intrapulmonary hemorrhage. This may become even more difficult if the sputum tests prove negative over a period of time (Fig. 12). Hemoptysis is common to both. Right angle projection serial laminagrams usually give direct information and frequently are conclusive. This nodular type of tuberculous infiltration, of course, lacks the communicating cord-like densities with the hilar structures. Frequently the laminagram reveals a cavity to indicate tuberculosis. Pulmonary

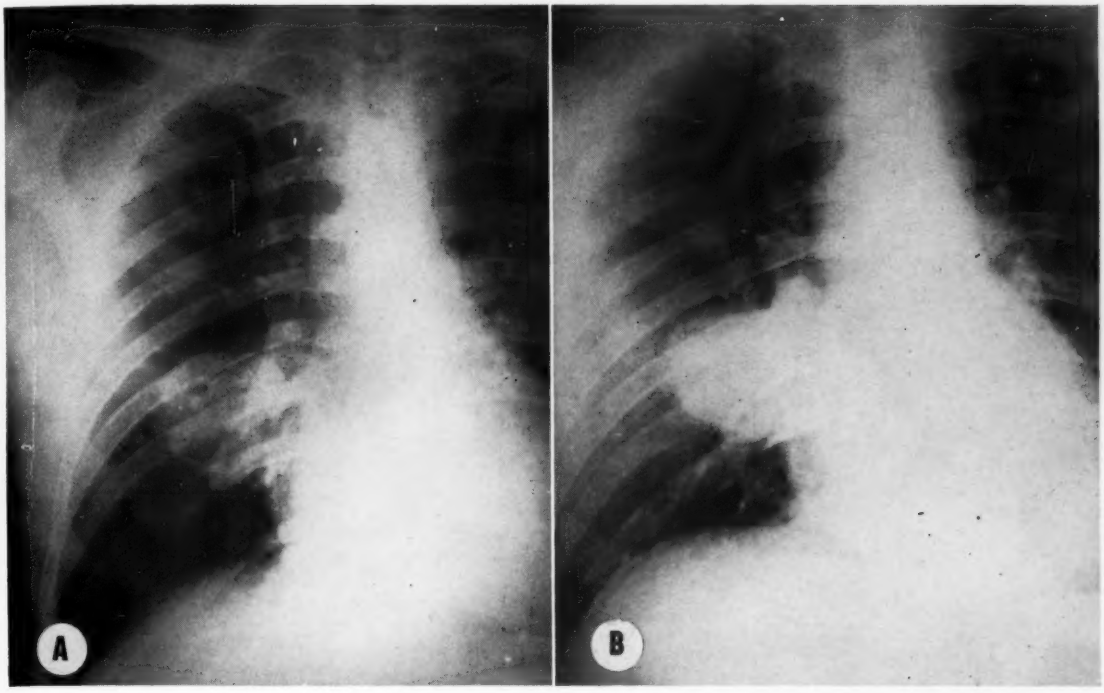

Fig. 7. Case VII. (*A*) The circular mass on the right represents an arteriovenous fistula. The dilated vessels leading to the hilar structures indicate the diagnosis. (*B*) Visualization of the heart and great vessels with 70 per cent diodrast demonstrates the contrast medium within the mass.

arterial aneurysms of a local type occur within tuberculous cavities—the so-called Rasmussen aneurysms. Positive sputum for tubercle bacilli readily differentiates these. Coccidioidomycosis likewise may offer a similar problem (Fig. 13). Often the skin test and the clinical findings are necessary to reach a diagnostic conclusion. Like the tuberculoma, the lesions calcify in time. This is never seen in a fistula.

Bronchogenic carcinoma, especially the alveolar cell type (Fig. 14*A*), may be difficult to differentiate from an arteriovenous fistula. Hemoptysis, cyanosis, and clubbing of the fingers and toes may be common to both. The carcinoma may appear as an obscure lung tumor which at times has a peribronchial extension from its body toward the hilus, giving the appearance of dilated vessels. The laminagrams are the final answer. No vascular connections with hilar structures are seen in the bronchogenic carcinoma. These lesions, like the tuberculoma, may eventually excavate and show widely disseminated flocculent infiltrative lesions in both lungs (Fig. 14*B*).

The mesothelioma (mesenchymoma) like coccidioidomycosis, because of the location near the chest wall, may require diagnostic pneumothorax to prove the extrapulmonary or intrapulmonary site respectively. The arteriovenous fistulas may have a similar appearance and location where pulsation is difficult to detect. Small cysts, particularly when they are multiple and closely approximated, are rarely confused with a vascular tumor or arteriovenous fistula. It must be remembered that these solid cysts may empty, usually are and stay small, and do not calcify. The connecting bands with the hilar shadows, common in the fistulas, are lacking in all of these.

Adenomas of the bronchus may show some resemblance to an arteriovenous fistula. Peribronchial extension from the mass may simulate communicating vessels

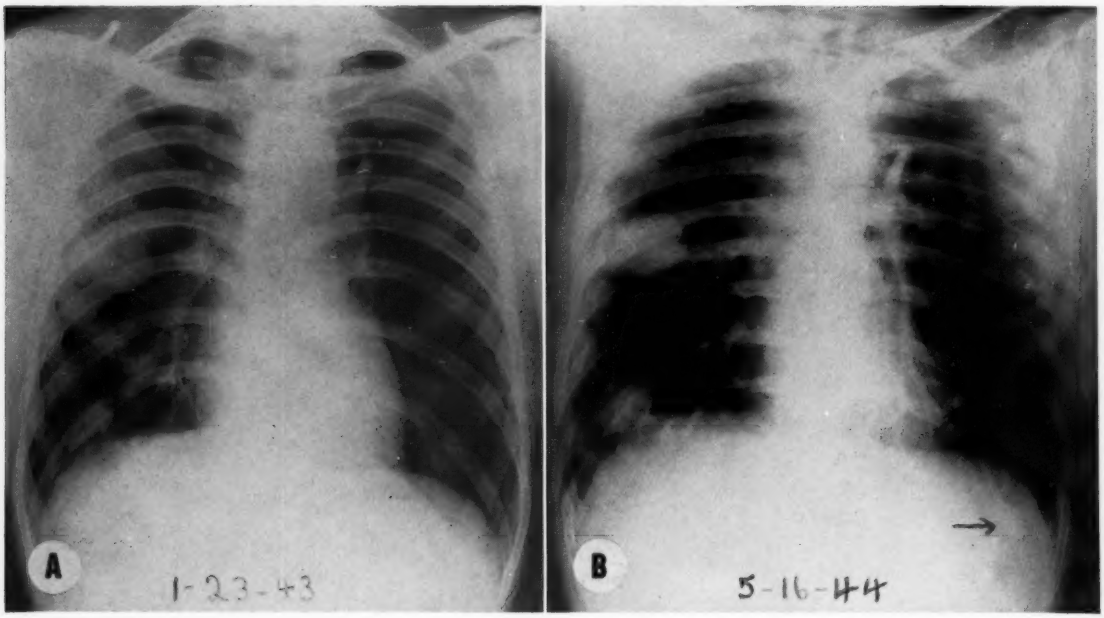

Fig. 8. (*A*) The vascular type of tumor and communications are demonstrated decisively by the angiocardiogram. Several fistulas are seen on the right. (*B*) The small opacities on the left were not suspected on the regular roentgenograms. (Courtesy Dr. J. E. Miller, Baylor University Hospital, Dallas.)

with the hilar structures. The metastatic lesions, particularly the sarcomatous type, are common in the lungs. In rare instances these are confused with the arteriovenous fistulas—more so when the primary malignant site is not found. Hemoptysis is common in all these. Laminagrams will disprove vascular connections with the hilar structures. A hernia of the diaphragm, either left or right, may be difficult to diagnose if not kept in mind. This may be confused with many other diseases, including arteriovenous fistula. The small hernia often representing an omental fat segment extending through the right hemidiaphragm may not have a bowel loop in its contents to help in identification. These are always seen anteriorly in the lateral projection and lack the usual cord-like bands leading to the hilar vessels (Fig. 9). Final diagnosis may have to be made, in some instances, at the operating table.

Hamartomas, which are benign tumors, often bilateral, formerly called chondromas, are believed to be of congenital origin, probably a malformation of a bronchial bud. These are sharply circumscribed and show central calcification which is typical. On cross section they show interesting bands of cartilage and epithelial lined clefts. They may be multiple or single. Serial laminagrams may bring out some of these characteristics which are never seen in the fistulas and the connecting bands with the hilar structures are lacking.

Intrapulmonary hemorrhage in arteriovenous fistulas, caused by rupture of a dilated vessel, may confuse the findings on the roentgenograms more often than expected. The primary lesion may be entirely obscured and the cardinal criteria for roentgenological diagnosis may be lacking because of this. In these instances the lesion may resemble many diseases, including unresolved pneumonia (Fig. 5), pulmonary infarct (Fig. 15), or bronchiectasis (Fig. 16).

It is important to stress that the clinical history of all these patients be made available to the radiologist. Likewise it is not hard to see why a certain small subpleural opacity representing an arteriovenous fis-

tula may escape roentgenological detection. It is unfortunate if the radiologist recognizes the lesion but is not surrounded by the teamwork for a complete clinical examination such as these patients need before final assessment to surgery. In extrapulmonary arteriovenous fistulas arteriography is usually the only means of demonstrating the lesion. The structure of the chest, however, provides so many different procedures that the roentgenological diagnosis should never be too difficult. The component parts of the chest are normally revealed in minute detail on roentgenological examination. This is largely due to the presence of air within the lungs which serves as a contrast medium, and the arteries, blood vessels and bronchi add to the variation in density of the structures in the chest wall and mediastinum. Any variation of these is usually discernible on a good quality roentgenogram.

CASE REPORTS

Case 1. A.M., an unmarried female, aged twenty, was admitted to the hospital on September 2, 1948, because of persistent cyanosis and chronic productive cough of several months' duration. The history revealed that she had had clubbing of the fingers and toes since birth. At the age of ten she was seen in the tuberculosis clinic because of cyanosis and contact with an active case of tuberculosis (father). A lesion in the right lung was found (Fig. 1*A*). The tuberculin test was positive and she was admitted to the City Tuberculosis Hospital. In August, 1948, a roentgenogram revealed the presence of an infiltrating process in the base of the right lung (Fig. 1*B*). Two subsequent specimens of sputum were reported as being positive for acid-fast bacilli.

Fig. 9. The right angle projection serial laminagrams were most helpful in this case. The opacity does not merge with communicating vessels. This proved to be a hernia of an omental fat pad through the right hemidiaphragm.

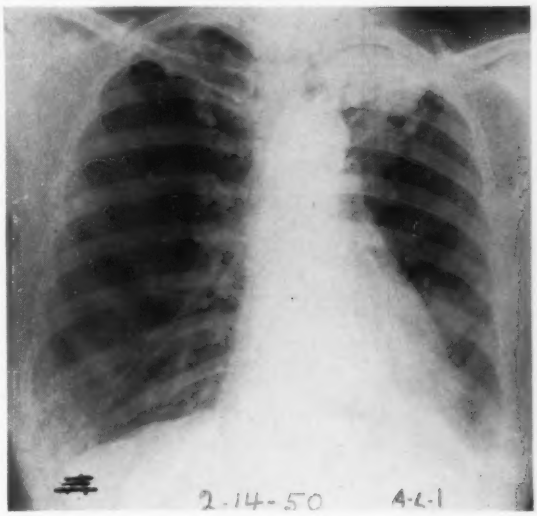

Fig. 10. The chain of lobulation on the left forms a configuration which is unusually large for tuberculomas. Segmental resection was done, and the pathological diagnosis was tuberculomas. The sputum was negative for acid-fast bacilli.

Family History. Father, fifty-four years of age, had active pulmonary tuberculosis until recently. No other members of the family have been cyanotic or have known congenital defects.

Physical examination at the time of admission revealed marked cyanosis and clubbing of the fingers and toes. The blood pressure was 118/74. The pulse rate was 120 per minute, the respiratory rate 26, and the temperature 99.6° F. Laboratory examination showed the red blood cell count to be 8.04 million and the hemoglobin 24 grams. The white blood cell count was 6,500, with an essentially normal differential count. The platelet count was 100,000 and the hematocrit 75 per cent. Bleed-

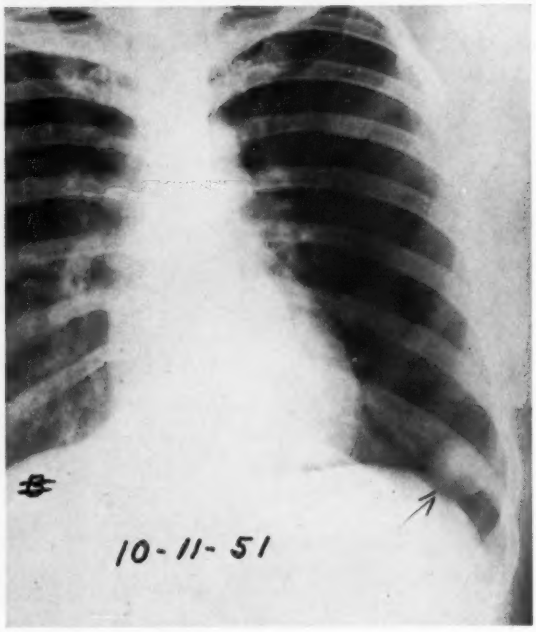

Fig. 11. The rounded tumor mass above the diaphragm on the left was an incidental finding in a forty-year old female. The opacity eventually showed central calcification, typical of a tuberculoma.

ing and clotting times were within the limits of normal. Kolmer and Kahn tests were negative. Four sputum examinations, taken on successive days following admission, for acid-fast bacilli, were negative, both on direct smear and concentration. On September 27, 1948, examination of a fresh specimen of sputum, following an episode of hemoptysis, revealed the presence of many acid-fast bacilli. Bronchoscopy soon after admission failed to reveal any evidence of abnormality. Heart catheterization performed on September 15 revealed the oxygen saturation of the samples obtained, as well as the pressures within the right auricle and ventricle, to be within the limits of normal. A bronchogram showed a lesion in the right lobe which prevented complete filling in the region of the posterior end of the lateral basal division of the right lower lobe bronchus. Several ribs on the right showed notching (Fig. 1*B*). The patient died on October 13, 1948, following a sudden massive hemoptysis.

Autopsy Report. There were some prominent tortuous vessels noted extending along the lower margins of the sixth and seventh ribs. The lungs (Fig. 1*C*) revealed two pulmonary arteriovenous communications, one in the middle and the other in the lower lobe.

Comment. One other case of arteriovenous fistula has been reported manifesting notching of the ribs. This unilateral notching cannot be explained except that there must have been undetected vascular communications between the lung circuit and the vessels of the chest wall, as in Case iv, herein reported. Liebow and his associates[4,5] have demonstrated that in chronic pulmonary disease and in a certain form of congenital heart disease there may develop an extensive collateral circulation and large anastomoses of the bronchial with the pulmonary arteries. This has thrown light upon many unanswered questions of the past but still does not give the explanation in this case because the collateral circulation was not prominent. This could, however, have been misjudged at autopsy because no special attention was focused on the ribs and intercostal vessels. This case also proved interesting and difficult to di-

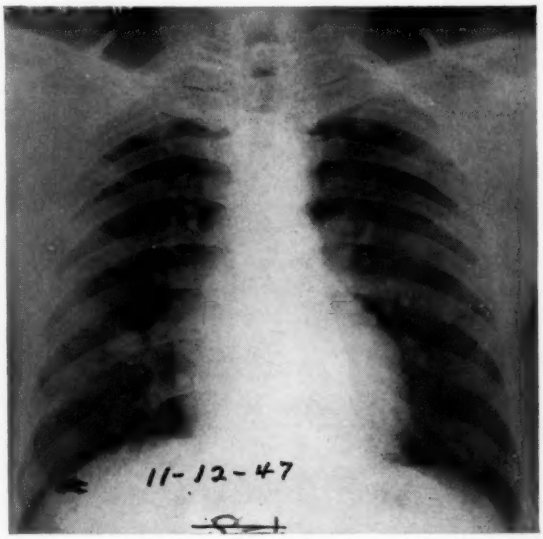

Fig. 12. Ball-shaped infiltration may be misdiagnosed even in the presence of many pulmonary calcified areas. This occurs especially in the presence of antibiotic therapy. The sputum cultures eventually were positive for acid-fast bacilli. A right lobectomy was done. The pathological diagnosis was pulmonary tuberculosis.

agnose because of the concurrent positive tuberculosis.

Case II. An American soldier, aged thirty-eight, had a chest roentgenogram because of cough and joint pain, at which time a left hilar mass was noted. Roentgenoscopy revealed marked expansile pulsation of the mass. Angiocardiography outlined the aneurysm wall but did not reveal any communicating cord-like bands. The clinical manifestations did not indicate a diagnosis of arteriovenous fistula; yet the roentgenological appearance was that of a large vascular tumor which appeared to have communicating vessels with the hilar structures. This was a pulmonary artery aneurysm (Fig. 2).

Case III. (Fig. 3) W. P. This man, aged twenty-eight, showed all the roentgenological criteria for the diagnosis of arteriovenous fistula which diagnosis was substantiated at operation. The large expansile mass in the lower lobe with at least two prominent cord-like bands connecting the hilar structures, and the increased density of the hilar region on the involved side are characteristic.

Case IV. E. J., male, white, aged thirteen, was seen by a local thoracic surgeon, Dr. J. E. Dailey, in consultation in January, 1948. The chief complaint of the patient was "coughing up blood." He stated that on January 24, 1947, while playing in the yard, he suddenly expectorated a large amount of blood. He was admitted to the hospital where he remained three months for observation for tuberculosis (Fig. 4*A*). However, all sputum examinations were negative. There was a history of dyspnea and cyanosis. He was discharged and went back to school, apparently well. On December 29, 1947, he again had severe hemoptysis which necessitated his receiving numerous blood transfusions. All sputum examinations were again reported as being negative for acid-fast bacilli. His past and family histories were not significant. No other member of the family had cyanosis or clubbing of the fingers and toes.

The physical examination revealed a very pale thirteen year old child, lying in bed and apparently quite ill. He was coughing and expectorating large quantities of bright red blood. Prominent arterial pulsations were noted just below and beneath the middle third of the left clavicle. A faint bruit was heard over this area but no thrill was felt. A loud to-and-fro murmur could be heard over the entire lower right chest from the spine posteriorly to the sternum anteriorly. The left lung was clear.

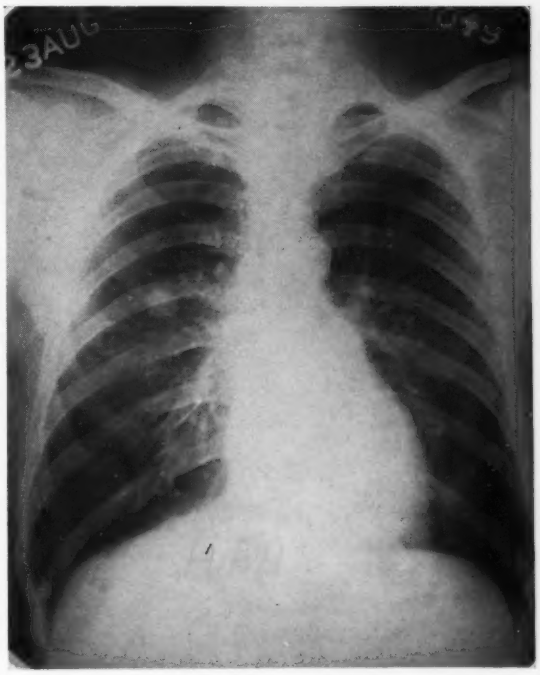

Fig. 13. This lesion represents coccidioidomycosis. Because of its location near the chest wall where pulsation is difficult to detect, this may be confused with many lesions, including arteriovenous fistula. Diagnostic pneumothorax may be required to prove its intrapulmonary location. Calcification common to the lesions disproves the fistulas even if the communicating bands are not discernible. The skin test and clinical symptoms may be the final proof.

The roentgenogram (Fig. 4*C*) showed marked increase in the vascular markings and several oblong densities in the right base communicating with dense hilar structures. Definite notching of the lower borders of the right eighth, ninth and tenth ribs posteriorly was noted. The heart was normal in size. There was no polycythemia because of repeated severe hemorrhage. The fingers and toes showed marked clubbing with moderate cyanosis. A diagnosis of hemangioma of the right lower lobe with arteriovenous fistula was made.

At operation on January 21, 1948, the right pleura was obliterated over the entire lower and middle lobes by highly vascular adhesions. Markedly large, tortuous vessels extending from the lung through the adhesions were noted anastomosing with the eight, ninth and

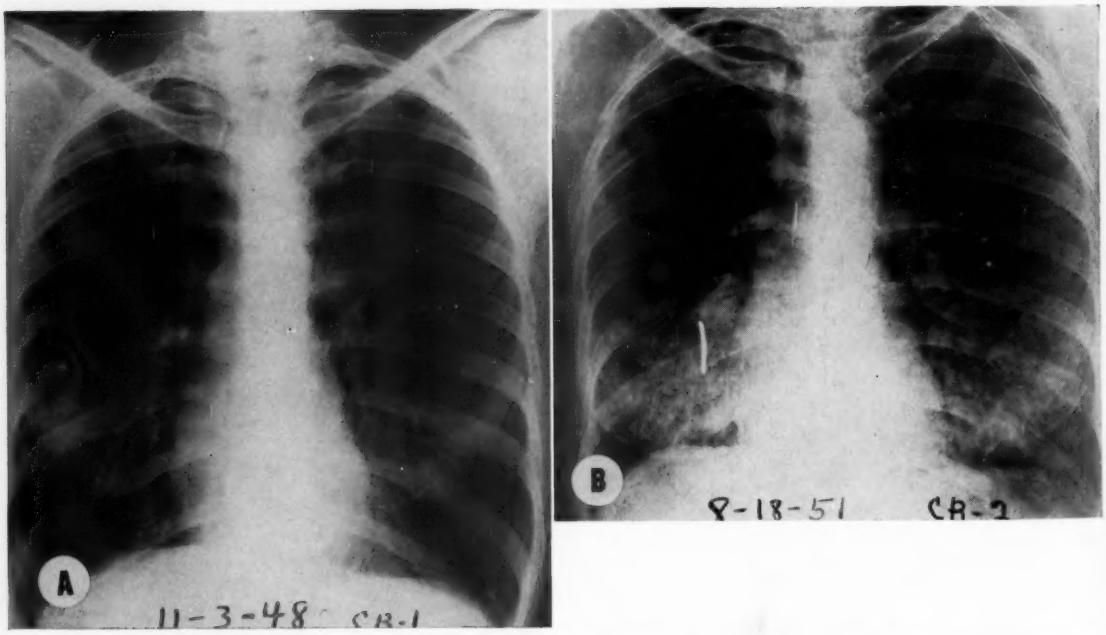

FIG. 14. (*A*) This small tumor mass on the right was found on routine physical examination. Suggestive peribronchial extension was seen in the serial roentgenograms simulating communicating bands with the hilar structures. (*B*) Indecision proved fatal. Autopsy revealed an alveolar cell carcinoma with metastatic spread to both lungs. Arteriovenous fistula was considered because of negative skin test and negative sputa for acid-fast bacilli.

FIG. 15. In rare instances a pulmonary infarct may be confused with arteriovenous fistula on the roentgenogram. Serial laminagrams should differentiate the fistula but were not done. Operation proved the lesion to be an infarct.

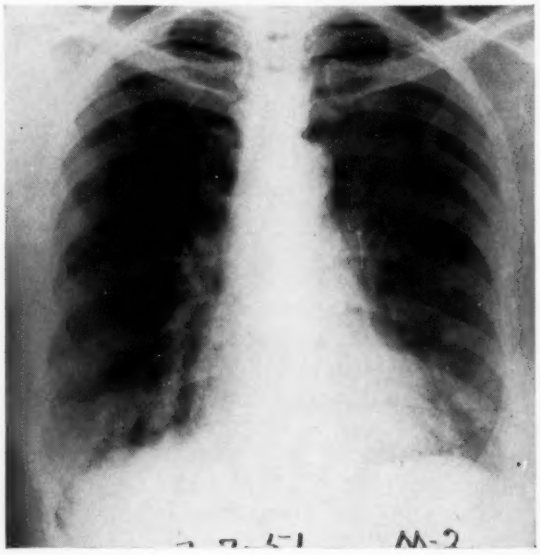

FIG. 16. Bronchiectasis associated with intrapulmonary hemorrhage, a common occurrence, will occasionally confuse the lesion enough to leave the diagnosis in doubt. Bronchograms may be required to be conclusive. Prominent bronchovascular shadows in the bases of the lungs represent dilated branches of the bronchial tree. The patient has clubbing of the fingers.

tenth intercostal vessels. These intercostal vessels were greatly enlarged and tortuous accounting for the notching of these ribs. The lower lobe was completely adherent to the diaphragm and huge vessels were seen extending from the lung into the diaphragm. Numerous very large, tortuous vessels could be felt throughout the entire lower portion, giving the impression of a mass of angleworms.

An incision was made then in the diaphragm and the subdiaphragmatic area was explored. The vascular channels were seen to go through the diaphragm and into the liver. Two or three areas in the liver could be palpated that gave the impression of hemangiomatous masses about the size of walnuts. The heart and great vessels appeared normal. The patient, following a right pneumonectomy, made an uneventful recovery and is living and well.

Postoperative diagnosis: Arteriovenous fistula of the right lower and middle lobes communicating with hemangiomas of the liver.

Case v. D. G., Latin American, male, aged forty-nine, who had the clinical triad of clubbing of the fingers and toes, cyanosis and polycythemia. A right pneumonectomy was performed by Dr. J. E. Dailey in July, 1947. The lesion in the right lung on the roentgenogram is not characteristic (Fig. 5). Similar shadows are produced by segmental atelectasis and unresolved pneumonia. This proved to be an arteriovenous fistula of the right upper and middle lobes. More frequently these fistulas are found in the lower lobes. The patient at present is well.

Case vi. (Fig. 6) J. S. This man, aged twenty-six, had the typical clinical criteria for the diagnosis of arteriovenous fistula of the right lower lobe, which was proved at operation. A lobectomy was done. The lesions were partly overshadowed by the cardiac silhouette and the diaphragm in the posteroanterior projection. In these locations the lesions, when small, may easily escape detection.

Case vii. (Fig. 7.) This man, aged twenty-six, complained of dizziness. He was in good health until sixteen years of age, when he had pneumonia, which was followed by persistent cyanosis. He had a typical Rendu-Osler-Weber syndrome. Roentgenological examination of the chest revealed a circular shadow of increased density in the right lower lung field which lay near the apex of the lower lobe. The inferolateral margin of this density showed a biconvex contour. On roentgenoscopy a slight pulsation of the pulmonic opacity could be seen. The oblique and lateral views showed a large vessel extending from the hilus and merging with the described shadow. The Valsalva test showed a slight decrease in the size of the opacity, and the Müller test showed an increase in size. Visualization of the heart and great vessels with 50 cc. of 70 per cent diodrast demonstrated the contrast medium within the mass. The roentgenological findings were consistent with an arteriovenous fistula.

A right total pneumonectomy was performed. All the telangiectasis of the face and mucous membranes disappeared within a year. After postoperative phlebothrombosis in the lower extremities he made an excellent recovery.

SUMMARY

Pulmonary arteriovenous fistulas have been reviewed briefly. The differential diagnosis has been dwelt upon because of the finding of these cases in places such as the tuberculosis hospitals and sanatoriums where they are often least suspected. Notching of the ribs in two cases herein reported is considered an incidental finding. Maier[9] found a similar condition in one of his cases. Hemangiomas of the liver coexisting and communicating with the pulmonary arteriovenous fistula in Case iv, herein reported, has merit. This condition has been suspected in a previously reported case[12] because of recurring anemia in the absence of cyanosis. Polycythemia was not discovered in the author's case because of extreme exsanguination. It might be concluded that the symptoms and signs are in proportion to the shunted blood supply. When there is more arterial than venous change the signs might be difficult to detect.

The value of certain techniques has been stressed. The roentgenological appearance alone should indicate the lesion but the diagnosis may be difficult in the presence of intrapulmonary hemorrhage. Lateral serial laminagrams give decisive information. Angiocardiograms usually give the final proof and should be employed routinely to

determine the number and location of the lesions before submitting the patient to surgery. A pneumonectomy is frequently necessary. For this reason knowledge of the condition of the contralateral lung is extremely important. In more than 50 per cent of the reported cases there have been two or more lesions.

1117 East Alabama
Houston 4, Texas

REFERENCES

1. BAER, S., BEHREND, A., and GOLDBURGH, H. L. Arteriovenous fistulas of lungs. *Circulation*, 1950, *1* (pt. 1), 602–612.
2. BIAMPALMO, A. Arteriovenous angiomatosis of lung with hypoxaemia. *Acta med. scandinav.*, 1950, *139*, Suppl. 248, 1–67.
3. CHURTON, T. Multiple aneurysm of pulmonary artery. *Brit. M. J.*, 1897, *1*, 1223.
4. LIEBOW, A. A., and others. Genesis and functional implications of collateral circulation of lungs. *Yale J. Biol. & Med.*, 1950, *22*, 637–650.
5. LIEBOW, A. A., and others. Studies on lung after ligation of pulmonary artery. *Am. J. Path.*, 1950, *26*, 177–195.
6. LINDGREN, E. Roentgen diagnosis of arteriovenous aneurysm of lung. *Acta radiol.*, 1946, *27*, 585–600.
7. MADDEN, J. F. Discussion. Telangiectasia (hereditary hemorrhagic)? Osler's "recurring epistaxis with multiple telangiectases of skin and mucous membranes"? *Arch. Dermat. & Syph.*, 1934, *30*, 295.
8. MAIER, H. C., HIMMELSTEIN, A., RILEY, R. L., and BUNIM, J. J. Arteriovenous fistula of lung. *J. Thoracic Surg.*, 1948, *17*, 13–26.
9. MAIER, H. C. Discussion. Pulmonary arteriovenous aneurysm. *Ann. Surg.*, 1950, *132*, 608–609.
10. MOYER, J. H., and ACKERMAN, A. J. Hereditary hemorrhagic telangiectasis associated with pulmonary arteriovenous fistula in 2 members of family. *Ann. Int. Med.*, 1948, *29*, 775–802.
11. READING, B. Case of congenital telangiectasis of lung complicated by brain abscess. *Texas State J. Med.*, 1932, *28*, 462–464.
12. ROBERTS, D. J., and HUTCHISON, J. E. Symptomless pulmonary arteriovenous aneurysms of fistulas. AM. J. ROENTGENOL. & RAD. THERAPY, 1951, *66*, 743–746.
13. RODES, C. B. Cavernous hemangiomas of lung with secondary polycythemia. *J.A.M.A.*, 1938, *110*, 1914–1915.
14. WILKENS, G. D. Ein Fall von multiplen Pulmonalisaneurysmen. *Beitr. z. Klin. d. Tuberk.*, 1917, *38*, 1–10.
15. YATER, W. M., FINNEGAN, J., and GIFFIN, H. M. Pulmonary arteriovenous fistula (varix). *J.A.M.A.*, 1949, *141*, 481–489.

SARCOIDOSIS*

AN ANALYSIS OF TWENTY-ONE PROVED CASES

By ALVIN W. FINESTONE, M.D
PARAMUS, NEW JERSEY

THIS paper presents a study of 21 proved cases of Boeck's sarcoid seen at the Jefferson Hospital, Philadelphia, during the five year period from 1947 to 1952.

Sarcoidosis is a generalized disease. It has an insidious onset and a predilection for lymphoid tissue. It runs a chronic relapsing course. Constitutional symptoms are few and, if present, are mild. Sarcoid most commonly occurs between the ages of twenty and thirty years. It may occur in any race, but it is of greater frequency in the colored and Scandinavian people. Table I illustrates the disposition of age, color, and sex in this series.

TABLE I

Age	Color	Sex
Range 22 to 50 yr.	White 29%	Male 29%
Average 30 yr.	Colored 71%	Female 71%

Pinner[8] considers the etiology to be that of atypical tuberculosis. Thomas[11] lists several authors who claim to have found tubercle bacilli in "sarcoid lesions." However, tuberculosis is not an infrequent complication of diabetes, Hodgkin's disease and leprosy. Other etiologies considered are virus, leprosy, Hodgkin's disease,[1] syphilis, mycoses, brucellosis, and inorganic agents such as beryllium and silica.[3]

The pathology is that of a miliary tubercle with a central giant cell surrounded by epithelial cells and lymphocytes. There is absence of caseation and calcification, and a tendency to undergo hyalinization with fibrosis.

Biopsy of the tonsils is frequently done in Scandinavia,[6] with a high percentage of positive results. Lung biopsy consisting of a small thoracotomy is being performed at Jefferson Hospital more frequently in diffuse pulmonary disease to obtain a more definitive diagnosis.

By examination of the tissues alone it may at times be impossible to differentiate sacroid from noncaseating tuberculosis, leprosy, mycoses, tularemia, lymphopathia venereum, syphilis, and berylliosis.

Table II and III show our patients' symptoms and signs.

TABLE II

SYMPTOMS IN 21 CASES

Dyspnea	9
Cough	8
Asymptomatic	4
Joint pains	4
Weight loss	4
Chest pain	2
Fatigue	1

TABLE III

SIGNS IN 21 CASES

Peripheral adenopathy	10
Skin	5
Hepatomegaly	4
Splenomegaly	1

These findings parallel those reported elsewhere in both asymptomatic and symptomatic groups.[7,9,10]

The literature shows that the majority of patients remain in good health.[5] Patients may succumb to sarcoid involvement of heart, heart failure secondary to diffuse pulmonary changes, and tuberculosis.

Our follow-up ranged from two weeks to five years, and the results are illustrated in Table IV.

* From the Department of Radiology, Jefferson Hospital, Philadelphia, Pennsylvania.

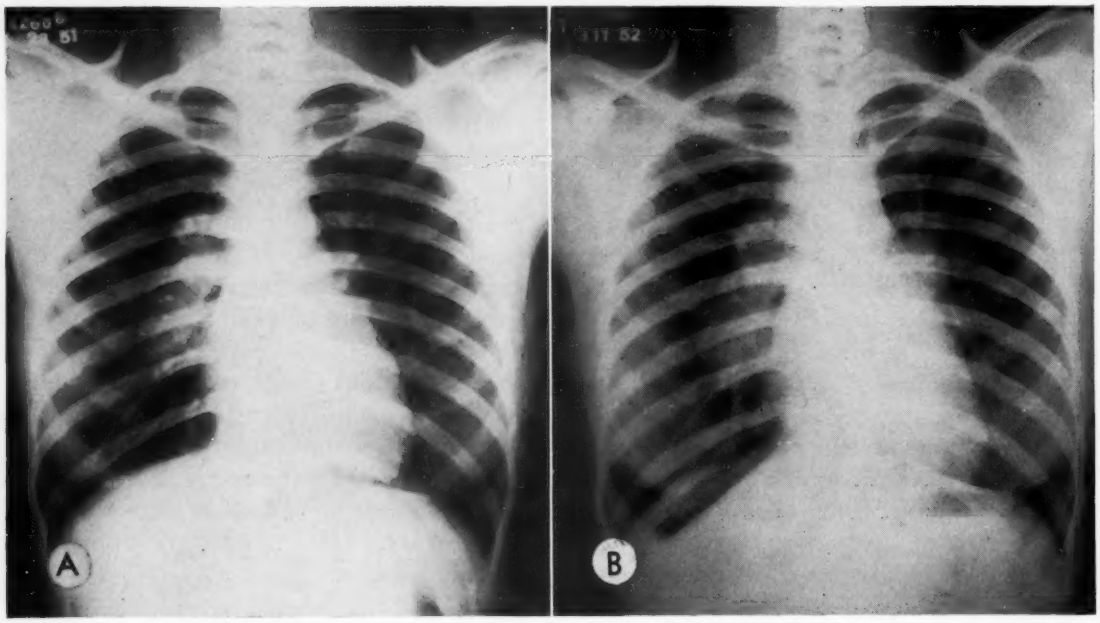

FIG. 1. M. D., colored female, aged twenty-two, with a history of joint pain and slight weight loss. Chest findings first noted on 70 mm. survey. Liver biopsy positive for sarcoid. (*A*) Posteroanterior view. Bilateral hilar and right paratracheal adenopathy. Lungs clear. (*B*) Posteroanterior view; one year later; no change.

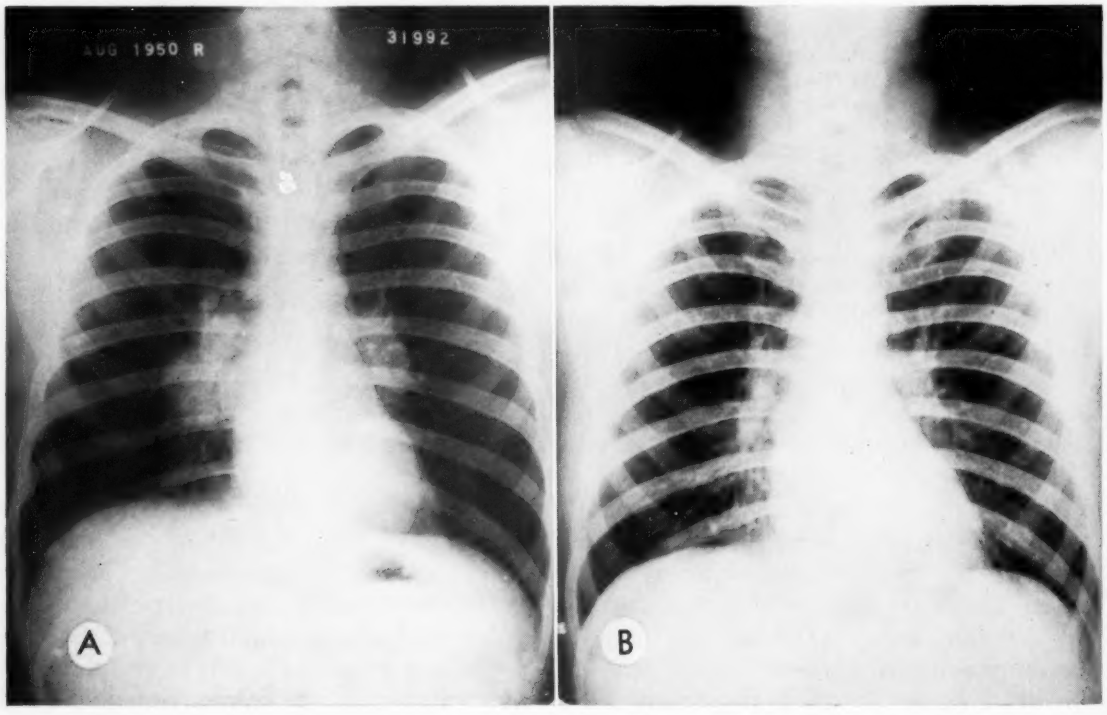

FIG. 2. A. F., white male, aged twenty-six, asymptomatic. Positive Kveim skin test. (*A*) Posteroanterior view. Bilateral hilar and right paratracheal adenopathy. Lungs clear. (*B*) Posteroanterior view. Three years later. Complete spontaneous regression of nodal disease.

TABLE IV

FOLLOW-UP IN 21 CASES

Good condition	11
Dyspnea	5
Dead	2*
Unimproved	2
Tuberculosis	1†

* Sarcoid a coincidental finding of histoplasmosis; cancer of gallbladder.
† Cavitation.

Table v is a summary of our laboratory findings.

TABLE V

SUMMARY OF LABORATORY FINDINGS

Study	Findings
Wassermann and Kline	1 in 21 positive
Red blood cells	2 in 21 anemia
White blood cells	2 in 21 leukopenia and leukocytosis
Total proteins	5 in 15 eight grams and over
Globulin	5 in 15 over three grams
PPD (strengths No. 1 and 2)	8 in 15 negative 2 in 15 slight positive 1 in 15 positive
Skins Tests	
Histoplasmin	0 in 10 positive
Coccidioidin	0 in 10 positive
Liver Function	
Cephalin flocculation	5 in 9 positive
Thymol turbidity	5 in 9 positive
Liver biopsy	8 in 8 positive
Sedimentation rate	7 in 10 elevated
Tubercle bacilli	1 in 8 positive

In comparing our laboratory results with those in the literature,[6,7,10] the findings were similar in regard to the tuberculin skin test, protein determinations, and liver biopsy. At Jefferson Hospital, the liver biopsy and liver function studies have been included in the studies. Eight cases in the present study had liver biopsy performed. All were diagnosed as sarcoidosis.

Several authors[3] have reported using the Kveim skin test with 93 per cent positive

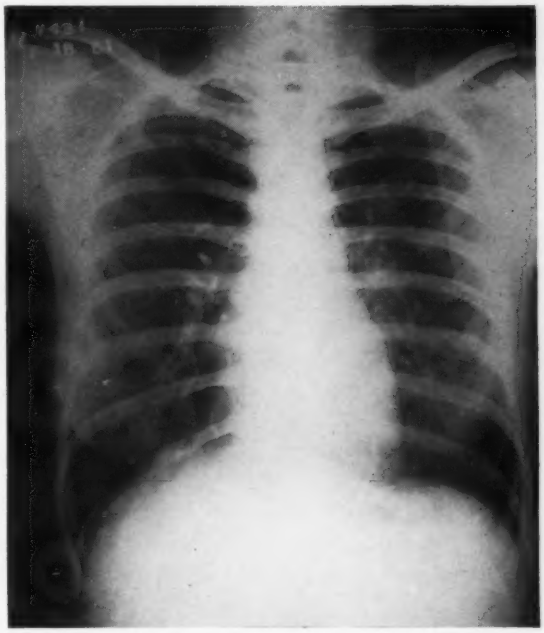

FIG. 3. C. B., white male, aged fifty, who was asymptomatic. Liver biopsy positive for sarcoid. Roentgenogram shows reticular and linear pattern of lungs.

results in active cases. At our institution this has not been employed. This test consists of an intradermal injection of the contents of a sarcoid node. After an interval of time, varying from approximately three weeks to several months, a red papular lesion may appear which persists for several months and on biopsy the histopathology is that of a granulomatous lesion consistent with sarcoid. Controls have been carried on in normals as well as in proved cases of tuberculosis, lymphopathia, and leprosy, and have been found to be negative in all.[2]

ROENTGEN FINDINGS IN THE PRESENT STUDY

Mediastinal nodes, alone, were involved in 35 per cent. Both the mediastinal nodes and lungs were involved in 40 per cent. The lungs, alone, showed positive changes in 25 per cent.

In approximately 50 per cent there was bilateral hilar and right paratracheal adenopathy. Bilateral hilar adenopathy was present alone in 25 per cent.

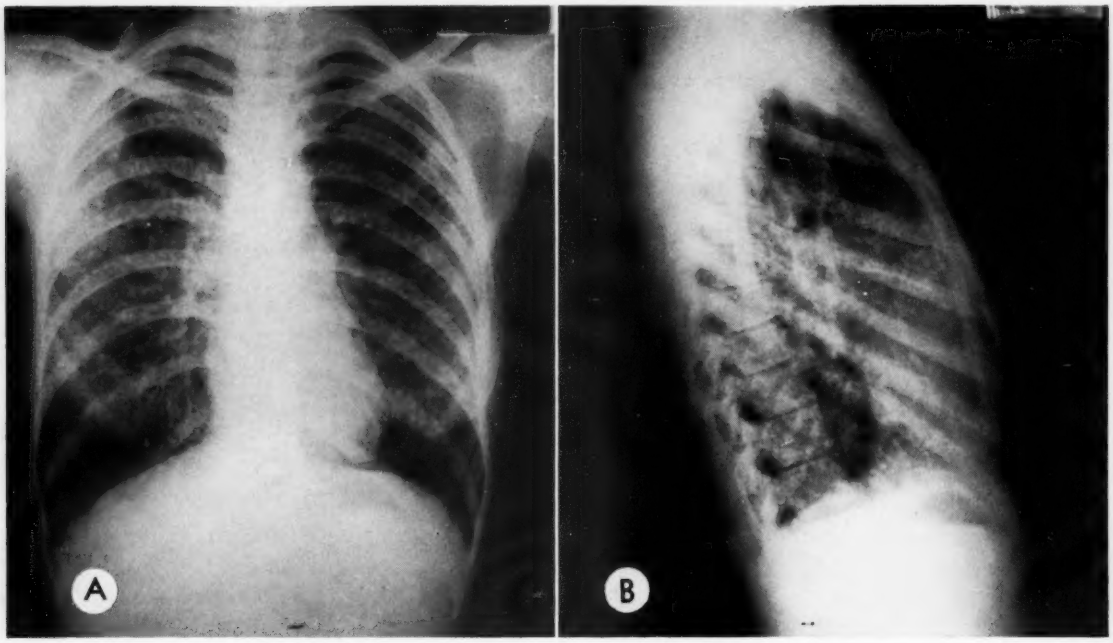

FIG. 4. E. T., colored female, aged thirty-two, with a history of cough and slight weight loss. Studies, including tuberculin skin test, negative for tuberculosis. Biopsy of liver positive for sarcoid. (*A*) Posteroanterior view. Diffuse miliary pattern of lungs. (*B*) Lateral view shows similar findings.

When the pulmonary changes were evaluated, there was a linear, strand-like pattern in almost 40 per cent. In another 40 per cent, nodular and/or confluent disease was found. A miliary pattern was found in 20 per cent.

There were 2 cases of unilateral hilar adenopathy and 2 cases of unilateral pleural effusion, both of which cleared spontaneously.

In regard to the distribution of the lung pathology, the apices were not significantly spared, but there was some predilection for the mid and lower lung fields. One case had predominant sided involvement and in another, the upper portions of the lungs were particularly involved.

If the clinical features of sarcoid are present, certain group patterns in the chest may help establish the diagnosis.

These are:

1. Bilateral hilar and right paratracheal nodes (with or without pulmonary involvement).[4]
2. Disseminated miliary and/or nodular densities without calcification in an asymptomatic case.
3. Massively enlarged hilar nodes in an asymptomatic patient.
4. Concomitant pulmonary disease with mediastinal nodes is much more common in sarcoid than in the lymphomas.
5. If laminagraphy is done, narrowing of the trachea or bronchi is usually not apparent.

DIFFERENTIAL DIAGNOSIS

The following are the various diseases which might be confused with sarcoid:

1. Lymphomas
2. Leukemias (especially chronic lymphatic)
3. Pneumoconioses
4. Pulmonary metastases (lymphatic and hematogenous)
5. Pulmonary congestion, second to cardiac failure.
6. Virus or coccal pneumonitis
7. Tuberculosis (miliary and fibrotic)
8. Mitral stenosis, with hemosiderin deposits

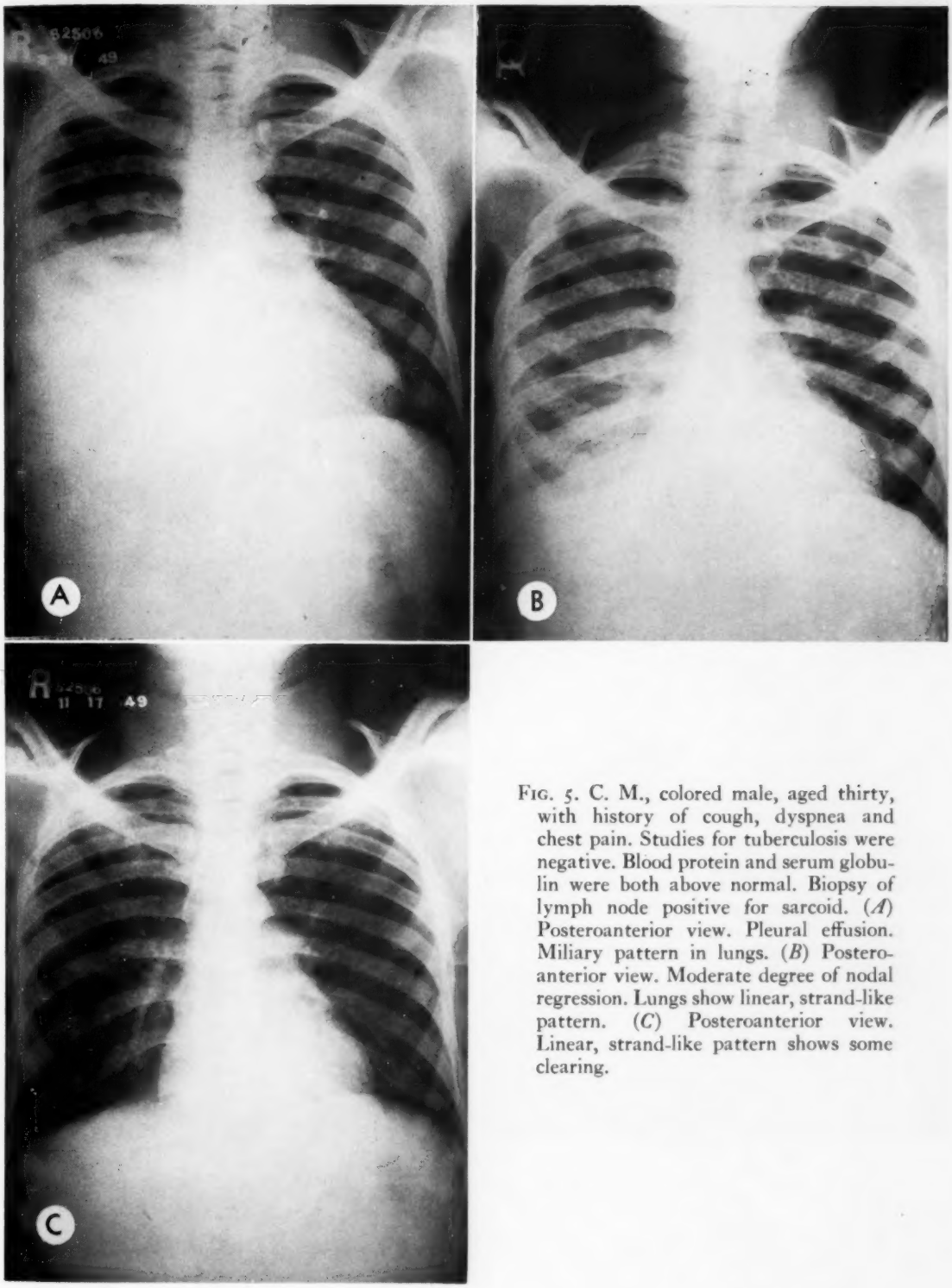

Fig. 5. C. M., colored male, aged thirty, with history of cough, dyspnea and chest pain. Studies for tuberculosis were negative. Blood protein and serum globulin were both above normal. Biopsy of lymph node positive for sarcoid. (*A*) Posteroanterior view. Pleural effusion. Miliary pattern in lungs. (*B*) Posteroanterior view. Moderate degree of nodal regression. Lungs show linear, strand-like pattern. (*C*) Posteroanterior view. Linear, strand-like pattern shows some clearing.

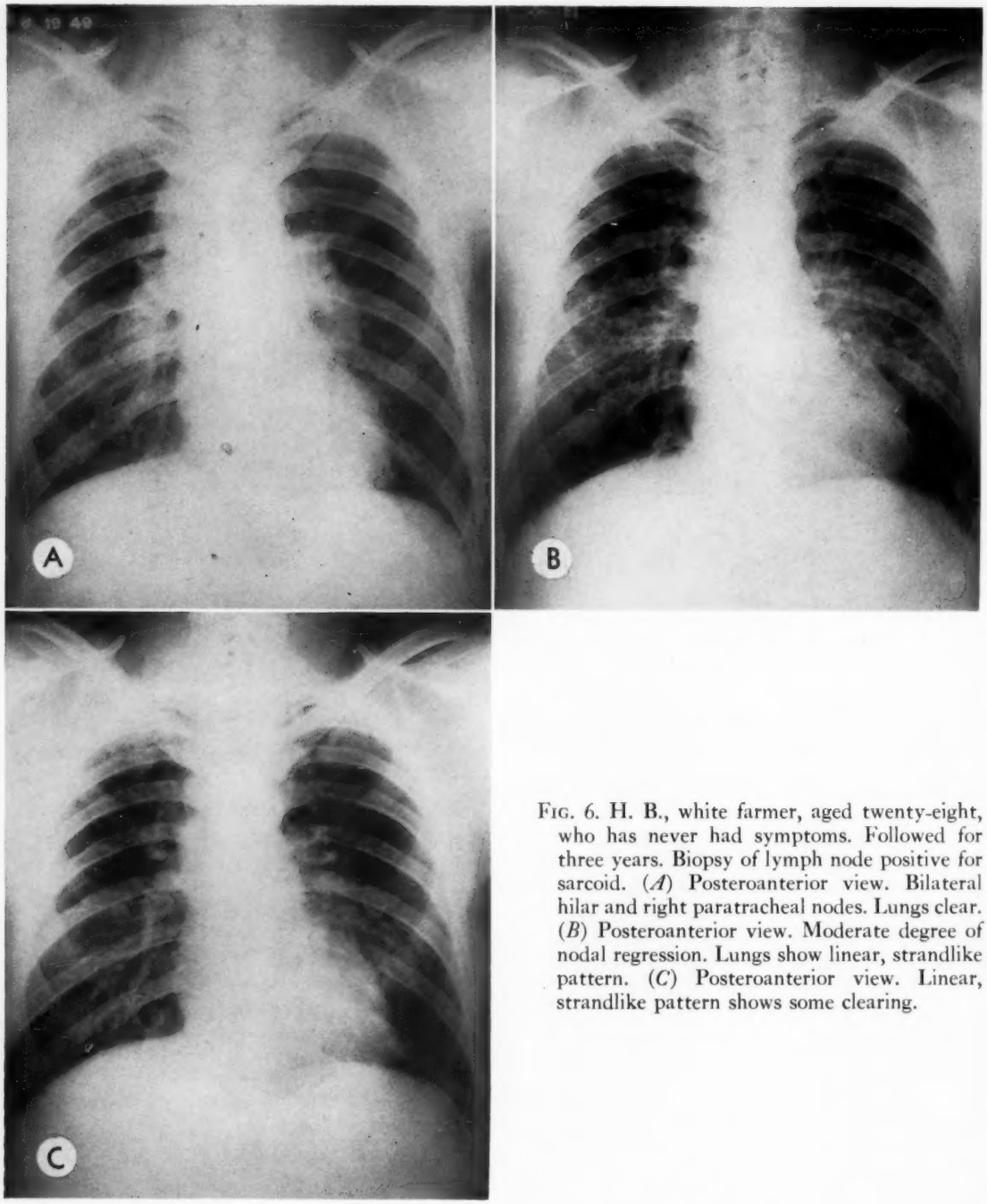

FIG. 6. H. B., white farmer, aged twenty-eight, who has never had symptoms. Followed for three years. Biopsy of lymph node positive for sarcoid. (*A*) Posteroanterior view. Bilateral hilar and right paratracheal nodes. Lungs clear. (*B*) Posteroanterior view. Moderate degree of nodal regression. Lungs show linear, strandlike pattern. (*C*) Posteroanterior view. Linear, strandlike pattern shows some clearing.

9. Fungus diseases
10. Loeffler's syndrome
11. Roentgen pneumonitis
12. Collagen diseases
13. Ayerza's disease

The lymphomas and chronic lymphatic leukemia will be analyzed briefly in comparison (Table VI). Several proved cases from Jefferson Hospital were picked at random.

In chronic lymphatic leukemia, 25 per cent showed intrathoracic findings. Of

Table VI

DIFFERENTIAL POINTS BETWEEN SARCOID, HODGKIN'S DISEASE, LYMPHOSARCOMA, AND CHRONIC LYMPHATIC LEUKEMIA IN THE PRESENT STUDY

Disease	Age in Years		Race		Sex		Splenomegaly	Chest Roentgenogram	Signs and Symptoms	Laboratory Findings
	Range	Av.	White	Neg.	M	F				
			Per Cent	Per Cent	Per Cent	Per Cent	Per Cent			
Sarcoid	22–50	30	29	71	29	71	Less 5	Frequent hilar and right paratracheal adenopathy Frequent combined nodes and lung pathology Mid lower lung fields more frequently involved Apices not significantly spared Spontaneous clearing and regression Miliary and nodular densities without calcification	19% asymptomatic Cough and dyspnea Skin lesions Generalized adenopathy Hempatomegaly or splenomegaly (unusual)	Normal count Protein elevated Globulin elevated Positive cephalin flocculation and thymol turbidity Increased sedimentation rate
Chronic lymphatic leukemia	37–73	60	88	12	75	25	88	Bihilar adenopathy and widening superior mediastinum The above, plus streaky densities in lungs Test irradiation; may get regression of leukemia in lungs when other parts of body are irradiated	Weakness Weight loss Bleeding Dyspnea Generalized adenopathy (90%) Hepatomegaly (50%) Splenomegaly (90%)	Leukocytosis (82–94% lymphocytes) Marked anemia increased sedimentation rate
Lymphosarcoma	16–68	43	100	0	69	31	31	Bihilar adenopathy and pleural effusion Widening superior mediastinum Lobulated hilar mass Test irradiation	Weight loss Dyspnea Joint pains Weakness Generalized adenopathy (50%) Palpable mass (90%) Hepatomegaly (40%) Splenomegaly (30%)	Moderate anemia, early Leukocytosis not significant Increased sedimentation rate
Hodgkin's disease	4–59	29	92	8	77	23	less 1	Bihilar and right paratracheal adenopathy with widening mediastinum Pleural and pericardial effusions Test irradiation	Cough Weight Loss Bleeding Pruritus Weakness Generalized adenopathy (40%) Hepatomegaly (40%) Splenomegaly (0%)	Moderate anemia, early Leukocytosis not significant

FIG. 7. R. H., colored female, aged thirty-one, with nodular lesions of lower extremities. Chest findings first noted on 70 mm. survey. Biopsy of skin nodule postive for sarcoid. Posteroanterior view shows predominantly right hilar adenopathy. Bilateral confluent densities in lower lung fields.

the cases of lymphosarcoma and Hodgkin's disease, there was chest involvement in 39 per cent and 31 per cent respectively.

Table VI summarizes the differential points between sarcoid, Hodgkin's disease, lymphosarcoma, and chronic lymphatic leukemia in the present study.

Patients with intrathoracic manifestations of the lymphomas and the leukemias are always relatively sick compared to those with the more benign sarcoidosis. The roentgen findings in themselves are not pathognomonic. In a high percentage of cases there is widening of the superior mediastinum. Pleural and pericardial effusions are more common. A hilar mass which may extend across half the chest or more is significant. A positive response to irradiation is quite helpful. When other parts of the body are irradiated, there may be regression of leukemic tumor in the lungs. There is usually no response in lymphosarcoma and Hodgkin's disease. It might be stated that when we have a clinical picture of tuberculosis with a lung mass, we should think of Hodgkin's disease. In pleural effusions of unknown etiology, a lymphoma should be ruled out.

Department of Radiology
Bergen Pines County Hospital
Paramus, New Jersey

I wish to thank Dr. Paul C. Swenson, Professor of Radiology, Jefferson Medical College Hospital, Philadelphia, Pennsylvania, for his help in the preparation of this paper.

REFERENCES

1. BADER, E. R. Sarcoid. *Radiology*, 1940, *35*, 482–485.
2. DANBOLT, N., and NILSSEN, R. W. Investigations on course of Kveim's reaction and its clinical value. *Acta dermat.-venereal.*, 1945, *25*, 489–502.
3. FREIMAN, D. G. Medical progress; sarcoidosis. *New England J. Med.*, 1948, *239*, 743–749.
4. GARLAND, L. H. Pulmonary sarcoidosis; early roentgen findings. *Radiology*, 1947, *48*, 333–352.
5. KING, D. S. Sarcoid disease as revealed in chest roentgenogram. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *45*, 505–512.
6. MCCORT, J. J., and others. Sarcoidosis. *Arch. Int. Med.*, 1947, *80*, 293–321.
7. MOYER, J. H., and ACKERMAN, A. J. Sarcoidosis. *Am. Rev. Tuberc.*, 1950, *61*, 299–322.
8. PINNER, M. Noncaseating tuberculosis; analysis of literature. *Am. Rev. Tuberc.*, 1938, *37*, 690–728.
9. REISNER, D. Boeck's sarcoid and systemic sarcoidosis (Besnier-Boeck-Schauman disease). *Am. Rev. Tuberc.*, 1944, *49*, 289–307.
10. RILEY, E. A. Boeck's sarcoid. *Am. Rev. Tuberc.*, 1950, *62*, 231–285.
11. THOMAS, C. C. Sarcoidosis. *Arch. Dermat. & Syph.*, 1943, *47*, 58–73.

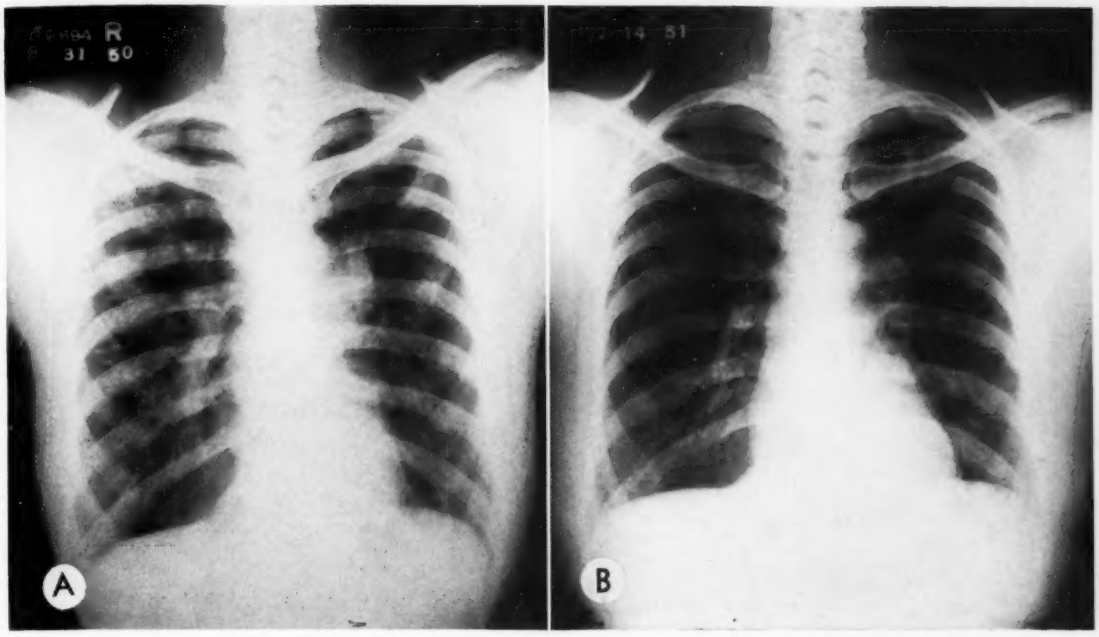

Fig. 8. W. M., colored female, aged twenty-eight, with a history of dyspnea and chest pain. Lymph node biopsy positive for sarcoid. (*A*) Posteroanterior view. Bilateral hilar and paratracheal adenopathy. Nodular densities seen throughout the lungs. (*B*) Posteroanterior view taken eighteen months later shows complete spontaneous regression of previous positive findings.

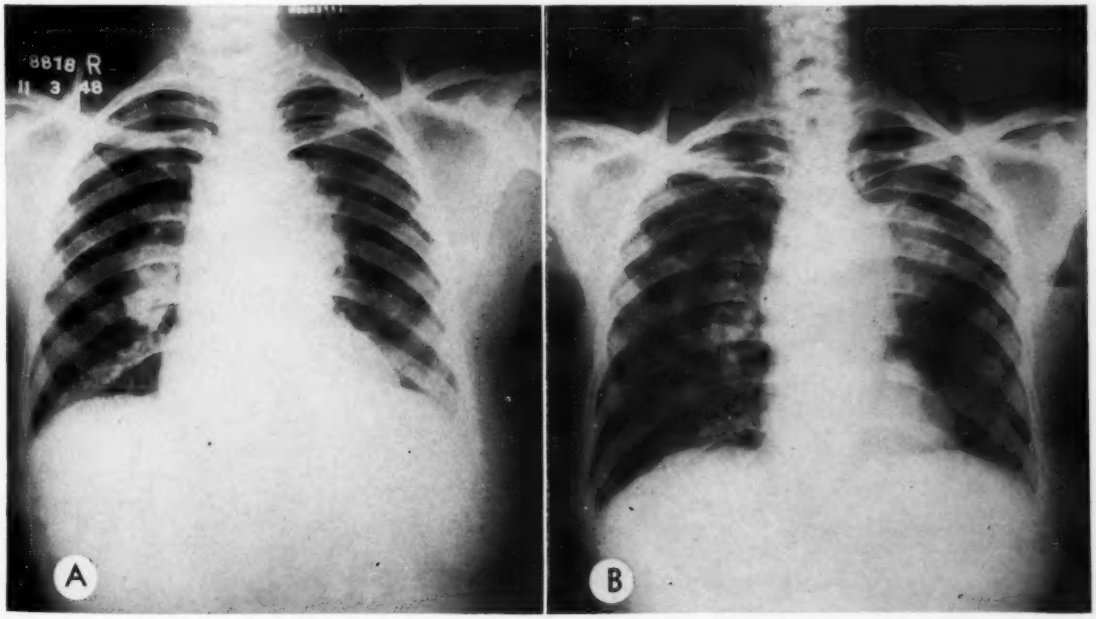

Fig. 9. M. H., colored female, aged fifty, in whom sarcoid was an incidental finding at autopsy. The patient had adenocarcinoma of gallbladder with metastases to the lungs. The skin, spleen and mediastinal lymph nodes were positive for sarcoid. (*A*) Posteroanterior view. Bilateral hilar and paratracheal adenopathy due to sarcoid. (*B*) Posteroanterior view. Two years later metastatic nodules are present in the lungs. The mass seen in the left hilus also represents metastasis.

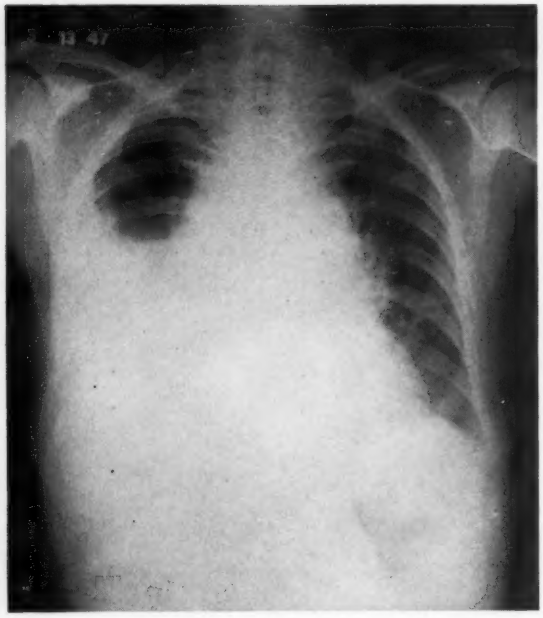

FIG. 10. C. K., white female, aged sixty, with a history of weakness and dyspnea. Blood studies diagnostic of chronic lymphatic leukemia. Posteroanterior view shows widening of superior mediastinum and bilateral hilar adenopathy; associated pleural effusion.

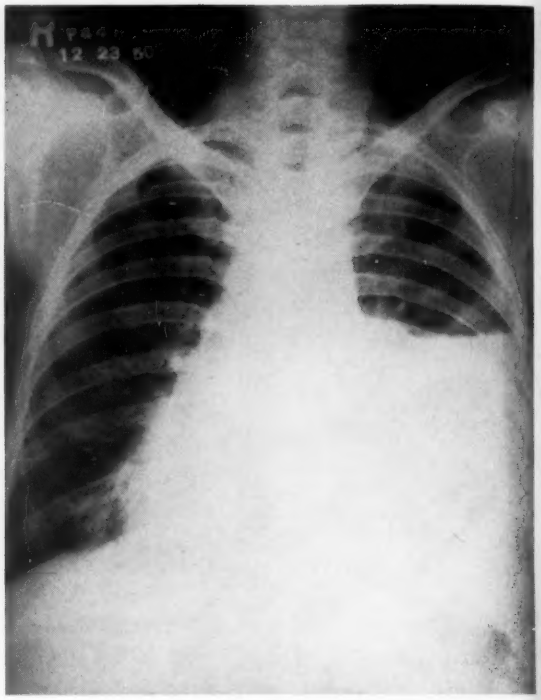

FIG. 12. J. C., white male, aged forty-two, with generalized adenopathy and swelling of the abdomen. Lymph node biopsy positive for Hodgkin's disease. Posteroanterior view shows pleural effusion and widening of superior mediastinum.

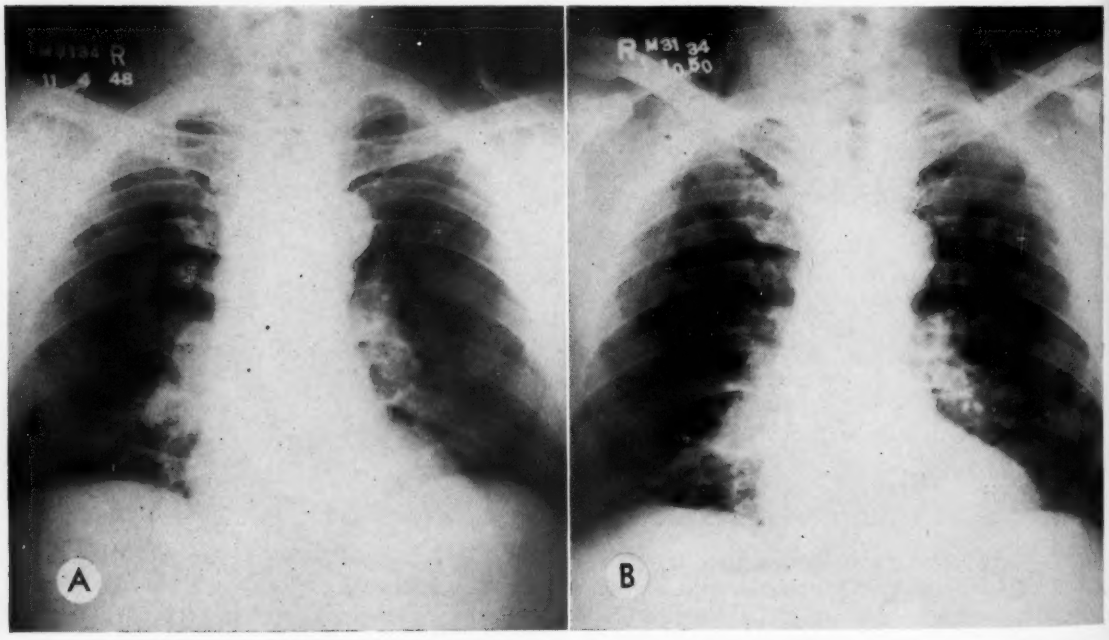

FIG. 11. C. A., white male, aged sixty-one, with history of dyspnea and swelling of the abdomen. General adenopathy and hepatomegaly present. Positive supraclavicular lymph node as well as positive findings in blood and bone marrow for chronic lymphatic leukemia. (*A*) Posteroanterior view shows bilateral lobulated hilar adenopathy and widening of superior mediastinum. (*B*) Posteroanterior view shows moderate regression following roentgen therapy.

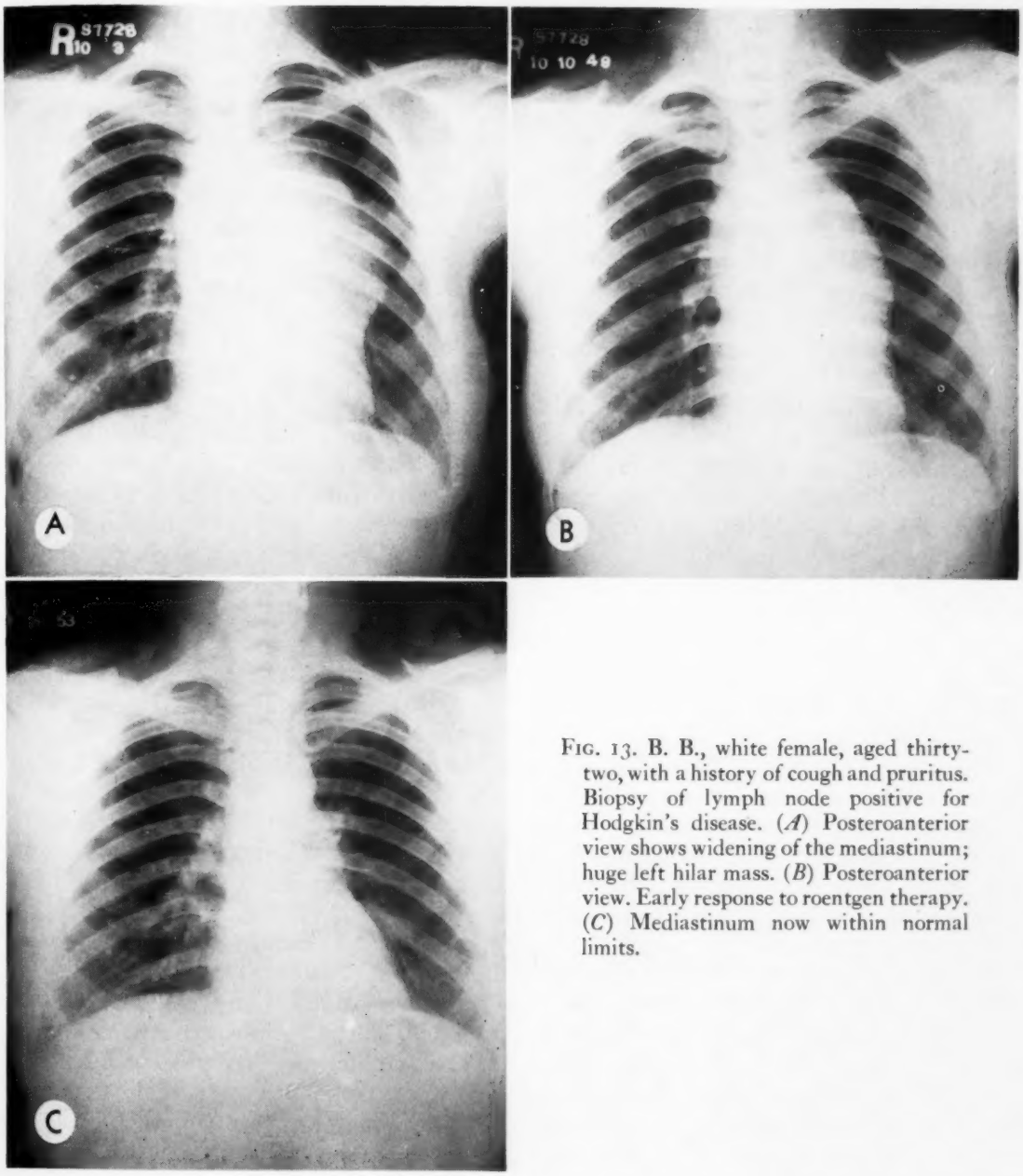

Fig. 13. B. B., white female, aged thirty-two, with a history of cough and pruritus. Biopsy of lymph node positive for Hodgkin's disease. (*A*) Posteroanterior view shows widening of the mediastinum; huge left hilar mass. (*B*) Posteroanterior view. Early response to roentgen therapy. (*C*) Mediastinum now within normal limits.

ROENTGENOLOGICAL OBSERVATIONS ON PLEURO-PULMONARY TULAREMIA*

By JOSEPH McK. IVIE, M.D.
NASHVILLE, TENNESSEE

DUE to the great advances made in chemotherapy in the last decade, specific treatment of certain types of pneumonia is now possible. Because of this, the roentgen differentiation as to etiology is becoming increasingly important. The following report is based on the roentgen study of 16 patients with pleuropulmonary tularemia. An attempt was made to determine if there was any characteristic feature in the conventional anteroposterior chest roentgenogram which would lead one to suspect the nature of the pneumonia.

Since the classic description of tularemia by Edward Francis,[7] much has been written about the disease. It occurs far more frequently than was formerly thought. Tularemia has been found in every state in the United States and Canada. The disease is transmitted to man by the handling of tissue of infected wild rabbits and other rodents, and through the bites of blood sucking ticks and flies.[10] The most important tick is the wood tick (*Dermacentor andersoni*), and the common dog tick (*Dermacentor variabilis*).[13]

The disease is usually divided into three clinical forms: (1) ulceroglandular; (2) oculoglandular, and (3) typhoidal types. Pneumonia may occur in any form. The portal of entry into the lungs is not always clear. Hunt[10] states that "the primary pulmonic form of tularemia as contrasted with pneumonia secondary to bacteremia and localization of *P. tularensis* in the lungs is probably rare." The incidence of pneumonia is probably much greater than is generally thought. Blackford and Casey[4] have emphasized the frequency and importance of pulmonary tularemia. Stuart and Pullen[15] found in their review of the reported cases that about 36 per cent of the patients with pulmonary tularemia had no primary lesion or demonstrable lymph node enlargement and therefore were classified as typhoidal types. Berson and Harwell[2] stress the difficult diagnostic problem of patients with tularemic pulmonary involvement in the absence of ulceroglandular lesions. In this series, 8 cases fall into the typhoidal types, and 8 were cases of pneumonia secondary to ulceroglandular tularemia. The fact that a large number of patients with tularemic pneumonia have no primary tularemic ulcer of the skin or mucous membrane, and that early clinical diagnosis of tularemic pneumonia is frequently difficult makes it very desirable to find some characteristic roentgen finding which would lead the physician to suspect *P. tularensis* as the etiological agent of the pneumonia. The clinician can then carry out other tests which will prove the diagnosis. This is particularly important since the mortality rate of untreated tularemic pneumonia is high,[6] and treatment by streptomycin is usually curative.[2,10,12]

P. tularensis was first shown to be highly susceptible to streptomycin by Heilman.[9] Foshay and Pasternack[6] reported the first case of human infection treated with streptomycin. Since then, there are many reports of cases treated with streptomycin with remarkable response.[2,5,10,12] It is generally accepted that streptomycin is the agent of choice in the treatment of patients with tularemia.

Sante[14] reported the first case of tularemic pneumonia demonstrated roentgenographically. In a study of the roentgen findings in pleuropulmonary tularemia, Bihss and Berland[3] describe two basic types. One group was manifested by hilar enlargement due to involvement of the tracheobronchial lymph nodes in the early stages of the disease with later extension

* From the Department of Radiology, Vanderbilt University Hospital, Nashville, Tennessee.
I am indebted to Thayer Veterans Hospital, Nashville, Tennessee, for data on patients in Veterans Administration Hospital.

into the parenchyma by retrograde spread via the lymph channels. The other group involved the parenchyma primarily, usually without enlarged hilar nodes. Archer *et al.*[1] found similar changes in the roentgen study of their cases. Although their roentgen findings were in accord with the known pathological findings, they felt the diagnosis could not be made from the chest roentgenogram alone.

In the present series, the records of 34 patients with tularemia treated at the Veterans Hospital were reviewed. Eleven (31 per cent) of these had definite pulmonary involvement and were classified as pleuropulmonary tularemia. The diagnosis was based on a rising serum agglutinin titer against *P. tularensis*. Serial 14 by 17 inch chest roentgenograms were made on each patient. The study of these, plus 5 cases of pleuropulmonary tularemia treated at Vanderbilt University Hospital, comprises the basis of this report.

It was interesting to note that in 9 of the 16 cases there was an oval area of infiltration seen on the first chest roentgenogram. This varied in its size and location. Its peculiar round appearance was similar to that of an abscess before the occurrence of cavitation. In one case, it resembled a large metastatic nodule. On one occasion, it was masked by pleural effusion, but could be seen following the removal of the fluid. This was the most constant and notable finding. In one case, a patchy area of density scattered throughout the left upper lobe was present. This was indistinguishable from pulmonary tuberculosis. In only 5 cases was hilar adenopathy a significant finding. In one of these cases, the diagnosis was suggested by one of the roentgenologists in the hospital from the chest roentgenogram alone. In 10 cases, the pleura was involved to some extent manifested by varying degrees of pleural effusion. In some instances, fluid was not seen in the pleural cavity on the first examination, but as the disease progressed, pleural effusion became manifest.

Although there was no one single sign that could be attributed to tularemia alone, it is felt that the common occurrence of an oval area of infiltration not usually seen in other pneumonias is a noteworthy finding. This seems to fit the known pathological findings[8,11] of a confluent lobular pneumonia with an appearance similar to that of caseous tuberculous pneumonia. This finding alone or in the presence of enlarged hilar lymph nodes should certainly lead one to consider the possibility of tularemia as the etiology of the pneumonia, although it is not pathognomonic of tularemia alone. In most cases, the presence of pleural fluid seemed to occur in relation to the length of the disease. It is felt that the presence or absence of pleural fluid will be of no special benefit in the differential diagnosis.

REPORT OF CASES

The following are brief summaries of illustrative cases.

Case I. H.C. This thirty-five year old white male entered the Veterans Hospital acutely ill, with the chief complaint of cough and pain in the left chest, chills and fever of five days' duration. He denied any contact with rabbits or ticks. He had a small lesion on his right hand, with a tender enlarged epitrochlar node. His temperature was 101.2° F., pulse 80, respiration 20 per minute, and blood pressure 120/60. On the day of admission, there was no serum agglutinin against *P. tularensis* present in the blood. Two weeks later, it was demonstrated in the serum in a dilution of 1:2560. The patient was started on streptomycin one week after admission, and showed dramatic improvement in temperature and general well-being. He was given 1 gram of streptomycin per day for thirteen days. His admission chest roentgenogram showed an oval area of density at the left base just above the diaphragm (Fig. 1). Five days later, there was a small amount of fluid at the left base. The patient's symptoms continued to improve, and three weeks after streptomycin treatment, the patient's chest roentgenogram was clear. He was discharged on the twenty-fifth hospital day as cured.

Case II. P.C. This twenty-eight year old white male, entered the Veterans Hospital with swelling and soreness of the right index finger, a large tender "kernel" in the right axilla, cough, chills, and fever of three days'

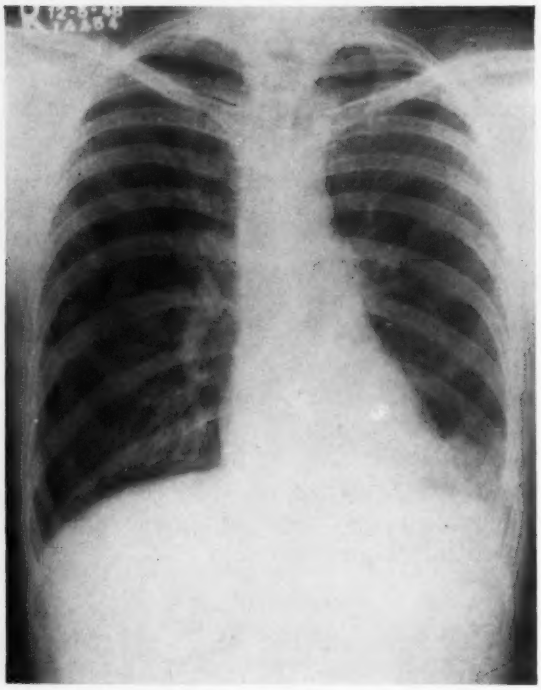

FIG. 1. Case I. There is an oval area of consolidation in the left lower lobe just above the diaphragm with slight pleural reaction.

duration. He had skinned a rabbit two weeks before admission. He had been given penicillin with no change in his condition. On physical examination, the temperature was 102.2° F., pulse 110, respiration 20 per minute, and blood pressure 132/65. The right index finger was swollen and there was an ulceration on the tip of the finger. The axillary lymph nodes on the right were enlarged and tender. There were faint sharp rales heard over the right lower chest only after coughing. On the day of admission, the serum agglutinins against *P. tularensis* were present in a dilution of 1:20, and seven days later the titer rose to 1:640.

He was started on streptomycin therapy, receiving a total of 12 grams in one week, with dramatic improvement in symptoms, and healing of the pneumonia and ulceroglandular lesions.

The admission chest roentgenogram showed an oval area of infiltration in the right cardiophrenic angle. There was patchy infiltration in the left base, with a small amount of fluid in the left costophrenic angle (Fig. 2).

CASE III. W.J. This thirty-two year old white male entered the Veterans Hospital complaining of high fever, chills, nausea, and vomiting of five days' duration. He had been treated with sulfadiazine and penicillin with no response. He had no history of contact with rabbits or ticks. His temperature was 105.8° F., pulse 120, and respiration 24 per minute. He appeared acutely ill and toxic. There were physical signs of fluid in the lower half of the right chest. There were no lesions on the skin and no enlarged nodes. The white blood cell count was 6,100. Blood agglutination with *P. tularensis* was negative. Because of the fact that tularemia was suspected, and because of the patient's serious condition, the was started on streptomycin treatment the second day of admission.

The first roentgenogram of the chest showed a large pleural effusion in the right base. A roentgenogram one day later, after removal of the fluid, showed a large oval area of infiltration in the right lower lobe which had the appearance of an unresolved abscess (Fig. 3). The patient had a very stormy course and the lesion was very slow in regressing. No agglutinins against *P. tularensis* were present in the serum until the forty-second day of admission. At that time, the serum agglutinin titer was 1:320, and it rose to 1:1280.

Comment. This case illustrates the seriousness and the real diagnostic problems

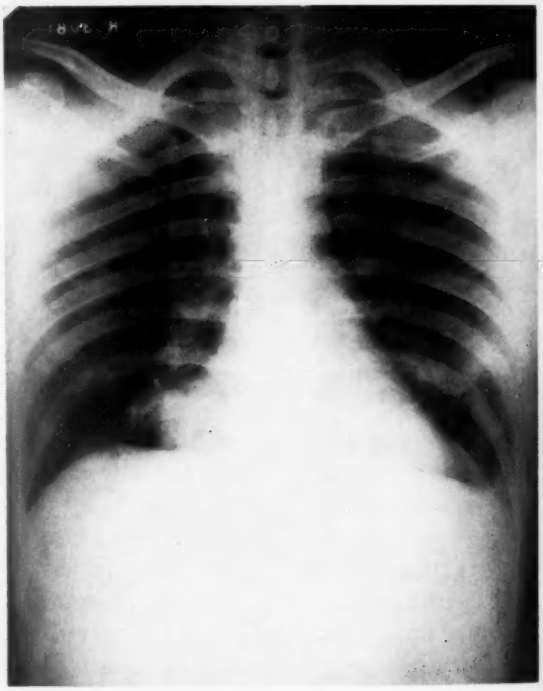

FIG. 2. Case II. There is an oval area of consolidation in the right cardiophrenic angle. There are patchy areas of infiltration in the left lower lung field.

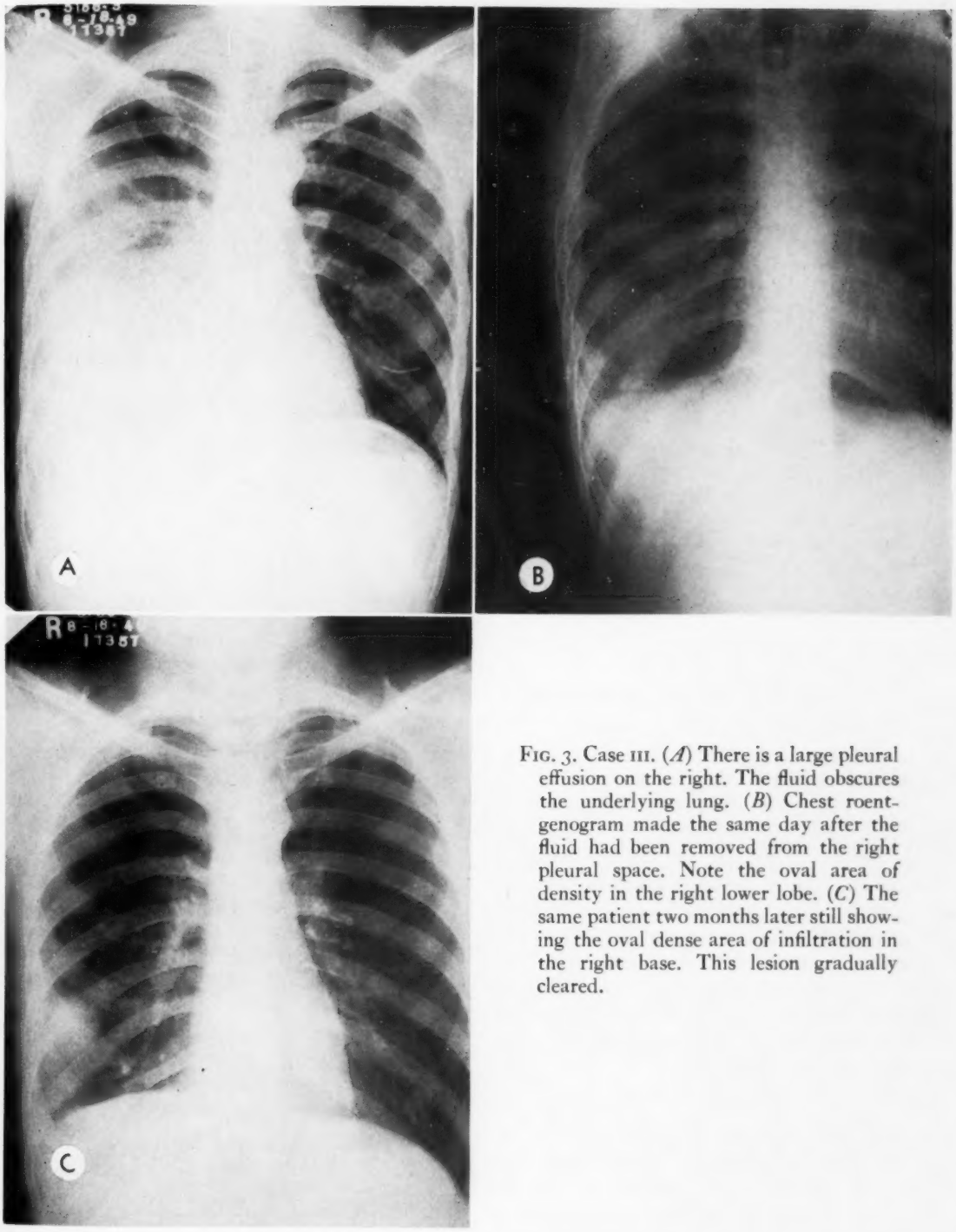

Fig. 3. Case III. (*A*) There is a large pleural effusion on the right. The fluid obscures the underlying lung. (*B*) Chest roentgenogram made the same day after the fluid had been removed from the right pleural space. Note the oval area of density in the right lower lobe. (*C*) The same patient two months later still showing the oval dense area of infiltration in the right base. This lesion gradually cleared.

this disease can create. It also shows the value of repeating the chest roentgenogram after the fluid has been removed. It is interesting to note the prolonged period of time before the appearance of serum agglutinins. The value of a therapeutic trial with streptomycin in suspected cases as suggested by Hunt[10] and Morgan[12] is obvious.

Case IV. J.M. This thirty-six year old white

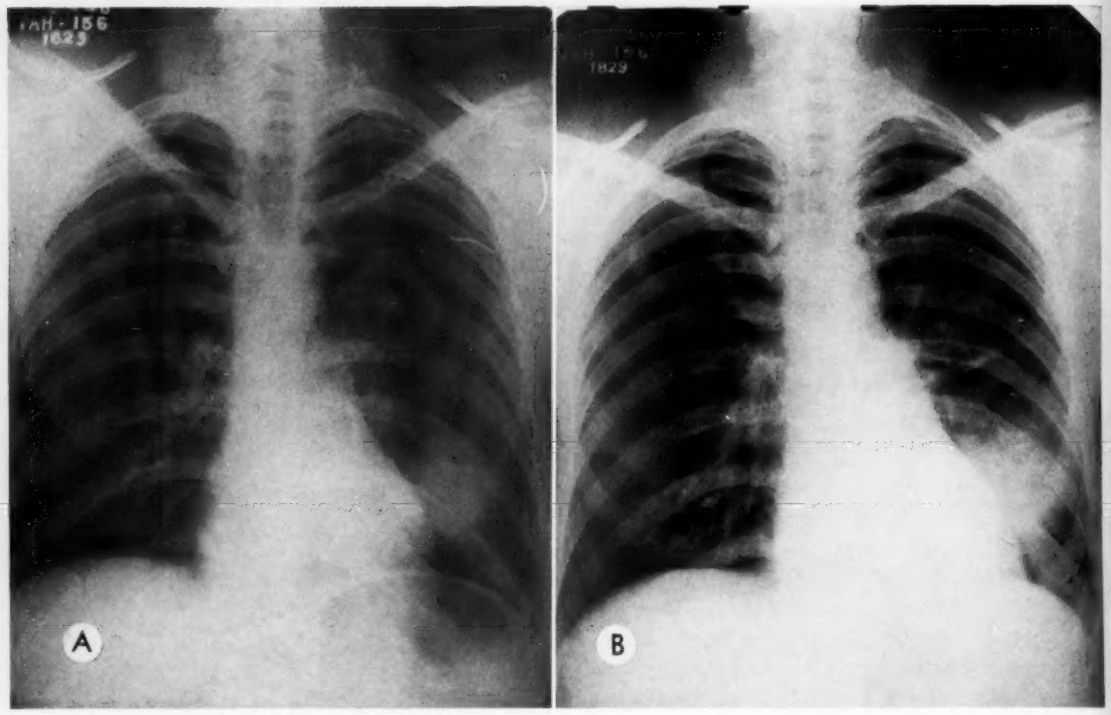

FIG. 4. Case IV. (*A*) The oval area of density is seen in the left lower lobe. (*B*) Chest roentgenogram three days later shows an increase in the infiltration. There is some thickening of the interlobar fissure. Four months after the initial chest roentgenogram the lung fields were clear.

male entered the Veterans Hospital with the chief complaint of chills, fever, pain in the chest, and cough of six days' duration. He had had no contact with rabbits. Previous to the onset of his illness, he had been working in the woods, and had pulled many ticks off his body with an occasional tick bit. On admission, he was acutely ill. His temperature was 103.8° F., pulse 88, respiration 28 per minute. There were signs of consolidation at the base of the left lung. The white blood cell count was 9,000. The roentgenogram on the day of admission showed an oval, dense area of consolidation in the left lower lung field (Fig. 4). The patient was treated with penicillin and sulfadiazine for two days with no improvement. On the second hospital day, the serum agglutinins against *P. tularensis* were present in a dilution of 1:160. He was then given streptomycin therapy and showed immediate and continued improvement. His serum agglutinin titer rose to 1:40,000. In spite of the clinical improvement, the roentgen findings remained constant for a long period of time, and a suggestive abscess cavity appeared in the oval area of infiltration. This gradually disappeared and the patient was discharged as cured.

CASE V. G.P. This sixty-two year old Negro male entered Vanderbilt University Hospital complaining of weakness and "pneumonia." Three weeks before admission, he developed general malaise, anorexia, weakness, and cough, which was later productive of sputum. He was told he had pneumonia, and he had been given penicillin and terramycin with no effect on the course of the disease. Physical examination showed the patient to be acutely ill, with generalized adenopathy. The temperature was 104° F., pulse 92, respiration 16 per minute, and blood pressure 140/80. The white blood cell count was 18,150.

On the admission roentgenogram, there was bilateral hilar adenopathy and patchy infiltration in both bases (Fig. 5). In consultation, the roentgenologist suggested that the findings were compatible with pulmonary tularemia. Further questioning of the patient revealed that three days before he became sick, he had been hunting, and had skinned a rabbit. Fol-

Fig. 5. Case v. There is enlargement of the hilar lymph nodes on both sides, with patchy areas of infiltration in the bases.

lowing this, he developed a small sore on his finger which had healed by the time of admission to the hospital. The serum agglutinins against *P. tularensis* were present in a dilution of 1:640. The patient was given streptomycin and improved rapidly.

SUMMARY

The roentgen findings in sixteen patients with pleuropulmonary tularemia have been reviewed. Eight of these patients with pulmonary tularemia had no evidence of ulceroglandular disease, and presented difficult diagnostic problems. All patients were treated with streptomycin therapy, and all recovered from the tularemia. While convalescing, one patient died suddenly from what was thought to be a pulmonary embolism. The findings in the posteroanterior roentgenogram of the chest varied and there is no one finding pathognomonic of tularemia. The high incidence of an oval lesion similar to an unresolved abscess was striking. This finding in a pneumonia, particularly one which did not respond to the usual therapeutic agents, should lead the roentgenologist to suspect and to suggest *P. tularensis* as the etiological agent of the pneumonia, so that proper therapy can be immediately instigated.

447 Doctors Building
Nashville 3, Tennessee

REFERENCES

1. Archer, V. W., Blackford, S. D., and Wissler, J. E. Pulmonary manifestations in human tularemia; roentgenologic study based on 34 unselected case. *J.A.M.A.*, 1935, *104*, 895–898.
2. Berson, R. C., and Harwell, A. B. Streptomycin in treatment of tularemia. *Am. J. M. Sc.*, 1948, *215*, 243–249.
3. Bihss, F. E., and Berland, H. L. Roentgenological manifestations of pleuropulmonary involvement in tularemia. *Radiology*, 1943, *41*, 431–437.
4. Blackford, S. D., and Casey, C. J. Pleuropulmonary tularemia. *Arch. Int. Med.*, 1941, *67*, 43–71.
5. Foshay, L. Diagnosis and treatment of tularemia. *Postgrad. Med.*, 1948, *4*, 315–321.
6. Foshay, L., and Pasternack, A. B. Streptomycin treatment of tularemia. *J.A.M.A.*, 1946, *130*, 393–398.
7. Francis, Edward. Tularemia; a new disease in man. *J.A.M.A.*, 1922, *78*, 1015–1018.
8. Goodpasture, E. W., and House, S. J. Pathologic anatomy of tularemia in man. *Am. J. Path.*, 1928, *4*, 213–226.
9. Heilman, F. R. Streptomycin in treatment of experimental tularemia. *Proc. Staff Meet., Mayo Clin.*, 1944, *19*, 533–559.
10. Hunt, J. S. Pleuropulmonary tularemia; observations on 12 cases treated with streptomycin. *Ann. Int. Med.*, 1947, *26*, 263–276.
11. MacCallum, W. G. Textbook of Pathology. Seventh edition, p. 580.
12. Morgan, H. J. Pleuropulmonary tularemia. *Ann. Int. Med.*, 1947, *27*, 519–528.
13. Parker, R. R., Francis, E., and Spencer, R. R. Tularemic infection in ticks of species *Dermacentor andersoni* (Stiles) in Bitter-Root Valley, Montana. *Pub. Health Rep.*, 1924, *39*, 1057–1073.
14. Sante, L. R. Pulmonary infection in tularemia; case report. Am. J. Roentgenol. & Rad. Therapy, 1931, *25*, 241–242.
15. Stuart, B. M., and Pullen, R. L. Tularemic pneumonia; review of American literature and report of 15 additional cases. *Am. J. M. Sc.*, 1945, *210*, 223–236.

COMBINED HIATUS HERNIA AND JEJUNAL DIVERTICULUM

REPORT OF A CASE AND REVIEW OF THE LITERATURE

By EDMUND W. KLINEFELTER, M.D.

YORK, PENNSYLVANIA

IT IS not well known among roentgenologists that diverticula of the jejunum may produce the same symptoms as hiatus hernia. In truth, the belief is common among them that jejunal diverticula are rarely, if ever, of clinical importance. Thus, if the jejunum is visualized during the barium meal examination and diverticula are seen, the roentgenologist frequently does not report them.[10] And again, if he does report them, he does so many times with the conviction that his report is of no significance other than to indicate that he has carefully examined this portion of the small intestine.[12] Too often the roentgenologist discontinues his barium meal examination as soon as stomach and duodenum have been explored so that jejunal diverticula do not have time to fill. Or if a five or six hour motor meal is employed, the diverticula most likely are empty. In any case, with the examiner following either one or both of the latter two procedures, jejunal diverticula are not visualized. Accordingly, with the preceding statements in mind, the paucity of case reports in the radiological literature on jejunal diverticula becomes understandable.

In contrast to the radiological literature on jejunal diverticula, that on hiatus hernia during the past decade has been abundant, so that most roentgenologists are well acquainted with the signs, symptoms and clinical importance of this condition.

Even as the radiological literature on jejunal diverticula is meager, that on the combination of hiatus hernia with jejunal diverticula is considerably more so. Some of the reports on hiatus hernia mention associated jejunal diverticula. Thus Simmons *et al.*[15] mention 3 cases of associated jejunal diverticula in 25 cases of hiatus hernia; Brick,[3] 2 cases in 308 and Polley,[11] 2 cases in 33. It should be noted, however, that in these cases of hiatus hernia, the studies were not made specifically for determining the presence of associated jejunal diverticula, and when this, together with the statements of the first paragraph, is considered, it can be understood why the above figures do not likely represent the true incidence. Indeed, the well known textbooks of diagnostic roentgenology by Golden,[7] Shanks,[14] Schinz[13] and Assmann[1] do not mention this combination. While the texts by Hodges[8] and Teschendorf[16] do contain illustrations of it, yet they give practically no details. A search of the literature, moreover, failed to reveal a single detailed case report of this combination of pathological conditions. The following representative case, therefore, is presented, and subsequently those features of it as well as those from the literature most likely of interest to the roentgenologist are outlined.

CASE REPORT

On January 1, 1951, a seventy-six year, 64 kg., 170 cm. white male shipping clerk was referred for roentgenologic examination. Over the past ten years he had been complaining at intervals of a feeling of pressure and discomfort in the left upper lateral umbilical region occurring ten to twenty minutes after meals. These attacks recurred approximately two to four times yearly. They were induced by too hastily eaten large bulky meals. They were relieved by avoidance of such meals. As long as he ate slowly and took six small nonbulky meals daily, he felt well. In his past history up to ten years ago, he never suffered any serious illness. At the age of forty-nine and again at fifty-two, following mild acute gastrointestinal upsets, barium meal examinations were done. The first examination showed no evidence of significant abnormality; while the second showed 2 small jejunal diverticula, but

nothing otherwise remarkable (Fig. 1 and 2). Eight months prior to the present examination, a slight degree of melena associated with discomfort in the left upper lateral umbilical region had been present over a five day period. And a similar episode, which had begun three days prior, was present during this examination.

Physical examination on January 20, 1951, revealed moderate tenderness to palpation in the left upper lateral umbilical region, but otherwise nothing significant. Laboratory studies revealed nothing of note other than a slight amount of blood in the stools and a hemoglobin of 85 per cent.

Barium meal examination on January 22 showed a 4 cm. hiatus hernia and eight small intestinal diverticula, with two of the latter located on the ascending duodenal loop, and the remaining six on the upper jejunum (Fig. 3 and 4). That portion of the small intestine from which the diverticula arose showed moderate dilatation and moderate thickening of the mucosal folds. Prior to the ingestion of the barium meal, palpation of the abdomen overlying the two largest diverticula revealed slight tenderness, but no masses. Shortly after ingestion of the meal when the herniated stomach became distended with barium, the patient did not experience a sense of pressure or discomfort, nor tenderness to palpation. When, however, the two largest jejunal diverticula became distended ten to twenty minutes after ingestion of the meal, he first experienced a sense of fullness and discomfort. At the same time the diverticula became considerably more tender. After a lapse of one to two hours when the diverticula had emptied, disappearance of the feeling of pressure and discomfort and decrease in the degree of tenderness were perceived. The small intestine distal to the upper jejunum, colon and gallbladder showed nothing remarkable on roentgenologic examination.

Fig. 1. Barium meal examination at the age of forty-nine shows no evidence of significant abnormality. There is no evidence of hiatus hernia or jejunal diverticulum.

Fig. 2. Barium meal examination at the age of fifty-two. There is no evidence of hiatus hernia. Two small diverticula are now present on an upper jejunal loop. They developed subsequent to the examination made three years before.

By January 30 the blood had disappeared from the stools and he was symptom free and remained so for a period of six months. From January 30, 1951, up to February 5, 1953, the time of the last examination, there were 4 episodes of the usual postprandial abdominal discomfort, but there was no melena.

ETIOLOGY

Age is regarded as the most important single etiologic factor in the development

FIG. 3. Barium meal examination at the age of seventy-six. Anteroposterior view. A hiatus hernia and six more diverticula have developed since the examination at the age of fifty-two. Note that the hernia is small and the lining mucosal folds normal. By roentgenoscopy it was reducible and not tender, and when distended produced no symptoms. The hernia was not causing symptoms.

of hiatus hernia and jejunal diverticula.[6,15] With increasing age there is a corresponding relative increase in the occurrence of these malformations, the greatest absolute incidence being between fifty and seventy years.[5] For the most part they are regarded as acquired rather than congenital.[4] With increasing age increasing degenerative changes take place in the tissues of the diaphragm and intestinal wall. These structures now become unable to withstand the stresses to which they are subjected and consequently abnormalities such as hernia and diverticula may develop. Thus, hiatus hernia and jejunal diverticula for the most part may be regarded as degenerative diseases and belong in the same etiologic group as arteriosclerosis and hypertrophic arthritis. Although these ideas concerning etiology represent those most widely accepted, a search of the literature failed to reveal a report of a case such as the one reported here actually depicting the development of this combination with increasing age.

PATHOLOGY

Hiatus hernia and jejunal diverticula may become acutely or chronically inflamed, may bleed, may ulcerate, may perforate and may form abscesses and adhesions.[10] Adhesions about the hiatus hernia may prevent spontaneous reduction of the hernia, while adhesions about jejunal diverticula may lead to acute or chronic intestinal obstruction. At times the intestine from which the diverticula arise may be dilated and show thick hypertrophied walls.[2,9]

SYMPTOMS AND DIAGNOSIS

Clinical. At least 20–30 per cent of the cases of this combination may be expected to show symptoms when each of their

FIG. 4. Barium meal examination at the age of seventy-six. Posteroanterior view. Note the two large jejunal diverticula protruding from the jejunum just beyond the duodenojejunal junction, and the thickened mucosal folds and widened caliber of the adjacent jejunum. By roentgenoscopy they were tender and when distended produced symptoms. The diverticula were causing symptoms.

diameters is over 3 cm.[3,4] In most cases, including mine, the symptoms produced by the hernia or diverticula are so closely similar to one another that clinical differentiation is impossible.[12] Thus in both conditions the most frequent clinical manifestations are: a feeling of pressure and discomfort or pain, sometimes colicky, in the upper abdomen coming on during or immediately after meals corresponding to the time when the sacs are distended with food. This pain may at times radiate to the back and scapula. Hurried eating of large bulky meals usually aggravates the discomfort. Slow eating of small nonbulky meals often serves to prevent it. At times postprandial discomfort may be relieved by maintaining a standing posture or be made worse by lying down. Tenderness to palpation in the epigastrium may be present. Symptoms of anemia may be present if there is bleeding. These symptoms tend to be intermittent and nonprogressive. Other less frequent appearing symptoms such as nausea and vomiting or regurgitation, belching, dysphagia, palpitation, anginal pain, dyspnea and cough occur more often with hiatus hernia, but also may be present with jejunal diverticula.[2,17]

Roentgenological. Since people are now tending more and more to live longer and since this combination of diseases appears increasingly with advancing years, the roentgenologist must expect to encounter it more often and should be on the watch for it. He has a special responsibility in those cases in which surgery is contemplated for hiatus hernia. In all these cases he must as far as possible make sure that the symptoms attributed to the hernia are not arising from an unsuspected jejunal diverticulum.[12] Although it is customary for roentgenologists to devote little or no attention to jejunal diverticula, it should be emphasized that at least 10–15 per cent of them do produce symptoms, and these symptoms can be differentiated from those produced by hiatus hernia only by careful roentgenoscopy during the barium meal examination.[2,10] Specific instructions, however, for making this differentiation could not be found in the literature. In view of this, such instructions are presented in the following paragraph.

The roentgenologist can determine the symptom producing hernia or diverticulum by noting: (1) Whether or not the patient develops a feeling of fulness and discomfort in the epigastrium when the hernial sac or diverticulum becomes distended with barium. Development of such symptoms early in the examination with distention of the hernial sac suggests that it is causative. Development of such symptoms later with distention of the diverticulum suggests the diverticulum. (2) Whether or not tenderness to palpation over the hernial sac or diverticulum is present. Tenderness over the hernial sac suggests that it is causative. Tenderness over the diverticulum suggests the diverticulum. (3) The thickness of the mucosal folds in the hernial sac. Thickened folds suggest that the hernia is causative. Normal folds suggest that it is not. (4) The reducibility of the hernia. Nonreducibility suggests that the hernia is causative. Reducibility suggests that it is not. (5) The thickness of the mucosal folds of the jejunum in the vicinity of the diverticulum. Thickened folds suggest that the diverticulum is causative. Normal folds suggest that it is not. (6) The caliber of the jejunum in the vicinity of the diverticulum. An increased caliber suggests that the diverticulum is causative. A normal caliber suggests that it is not. (7) The size of the hernial sac or diverticulum. The larger, the more likely they are causative. The smaller, the less likely.

The application of these instructions is exemplified by the case reported here. In this case, no symptoms were experienced when the hernial sac became distended, there was no tenderness over the hernia nor thickening of mucosal folds, and the hernia was small and reducible. The hernia was symptomless. On the other hand, later in the examination when the two largest jejunal diverticula became distended, symptoms developed; sharply localized tender-

ness over the diverticula was present, and the jejunum adjacent to the diverticula showed moderately widened mucosal folds and increase in caliber. The diverticula were causing symptoms.

TREATMENT

In the presence of symptoms, the most important single factor in treatment is avoidance of overloading of the upper gastrointestinal tract with food.[15] Six small nonbulky meals should be taken daily instead of three. Food should be thoroughly chewed and eaten slowly. The second most important factor is avoidance of increase of intra-abdominal pressure such as may result from constipation, heavy lifting or stooping. Maintenance of the upright posture after meals may at times be of benefit. Occasionally sedative and relaxing drugs such as phenobarbital and belladonna afford some relief. Most patients, just as mine, may be satisfactorily treated by these measures.[11] In a few cases, however, if the symptoms become unbearable or there is serious bleeding, surgical repair of the hernia or excision of the portion of the jejunum bearing the diverticula must be considered.

SUMMARY

1. With increase in the length of life the roentgenologist must correspondingly expect to encounter more and more cases of combined hiatus hernia and jejunal diverticula.

2. A representative case of this combination of conditions is presented, and those features of it as well as those from the literature with which the roentgenologist should be familiar are outlined.

3. The roentgenologist must remember that symptoms resulting from hiatus hernia cannot be differentiated clinically from those due to jejunal diverticula.

4. Differentiation of the symptom-producing hiatus hernia from the symptom-producing jejunal diverticula can be made only during the barium meal roentgenoscopic examination. Instructions for making this differentiation are given.

546 West Market St.
York, Pennsylvania

REFERENCES

1. ASSMANN, H. Die klinische Röntgendiagnostik der inneren Erkrankungen. J. Springer Verlag, Berlin, 1950, vol. 2, p. 418.
2. BENSON, R. E., DIXON, C. F., and WAUGH, J. M. Nonmeckelian diverticula of jejunum and ileum. *Ann. Surg.*, 1943, *118*, 377–393.
3. BRICK, I. B. Incidence of hiatus hernia and associated lesions diagnosed by roentgen ray. *Arch. Surg.*, 1949, *58*, 419–427.
4. EISEN, D. Oesophageal hiatus hernia (with special reference to its x-ray diagnosis). *Canad. M.A.J.*, 1938, *39*, 207–213.
5. GILBERT, N. C., DEY, F. L., and RALL, J. E. Recurrent hiatus hernia. *J.A.M.A.*, 1946, *132*, 132–134.
6. GISBERTZ, H. Multiple Dünndarmdivertikel. *Arch. f. klin. Chir.*, 1931, *165*, 314–321.
7. GOLDEN, R., Editor. Diagnostic Roentgenology. Williams & Wilkins Co., Baltimore, 1952, vol. 1, p. 265.
8. HODGES, F. J. The Gastro-Intestinal Tract. Year Book Publishers, Chicago, 1944, p. 313.
9. KUHLMANN, F. Der Dünndarm im Röntgenbild. Urban & Schwarzenberg, Berlin, 1951, p. 78.
10. ORR, I. M., and RUSSELL, J. Y. W. Diverticulosis of jejunum. *Brit. J. Surg.*, 1951, *39*, 139–147.
11. POLLEY, H. F. Congenital short esophagus with thoracic stomach and esophageal hiatus hernias. *J.A.M.A.*, 1941, *116*, 821–825.
12. POPMA, A. M., and STONE, A. M. Hiatus hernia and gastrointestinal diverticulosis. *Northwest. Med.*, 1951, *50*, 773–774.
13. SCHINZ, H. R., BAENSCH, W., and FRIEDL, E. Lehrbuch der Röntgendiagnostik. G. Thieme, Leipzig, 1939, vol. 2, p. 411.
14. SHANKS, S. C., KERLEY, PETER, and TWINING, E. W., Editors. A Text-Book of X-Ray Diagnosis. H. K. Lewis & Co., London, 1938, vol. 2, p. 181.
15. SIMMONS, E. E., LONG, R. S., HUNT, H. B., and MOORE, R. C. Hiatus hernia. *Arch. Int. Med.*, 1950, *86*, 253–265.
16. TESCHENDORF, W. Lehrbuch der röntgenologischen Differentialdiagnostik. G. Thieme, Stuttgart, 1950, vol. 2, p. 3.
17. WILLIAMS, C., and WALKER, J. B. Diverticula of jejunum. *Virginia M. Monthly*, 1946, *73*, 212–215.

Vol. 74, No. 3

ROENTGEN DIAGNOSIS OF ESOPHAGEAL VARICES*

COMPARISON OF ROENTGEN AND ESOPHAGOSCOPIC FINDINGS IN 502 CASES

By I. E. KIRSH, M.D.,† C. C. BLACKWELL, M.D.,‡ *and* H. D. BENNETT, M.D.§

From the Departments of Radiology and Internal Medicine, Veterans Administration Hospital

HINES, ILLINOIS

VARICES develop in the esophagus as a result of obstruction to the normal downward flow of blood in the esophageal veins. This obstruction may be due to thrombosis or tumor within the venous system itself including the coronary veins of the stomach, splenic vein and portal vein, or much more commonly due to obstruction within the hepatic bed as a result of liver disease with increase in portal vein pressure.[10]

The importance of esophageal varices clinically lies solely in the fact that they may bleed, and bleed dramatically. Hemorrhage from bleeding esophageal varices has been estimated to cause between one-quarter and one-third of all deaths due to cirrhosis of the liver.[3,11]

Prior to fifteen years ago the diagnosis of esophageal varices was of importance only in the sense that such a diagnosis told the physician there was nothing more that could be done for the patient. Since that time, however, two major developments have occurred. Esophageal compressive tubes such as the Sengstaken tube[2,16] permit the emergency treatment of bleeding esophageal varices. As a result of the development of various surgical procedures, including esophagogastrectomy,[15] splenectomy, splenorenal and portocaval anastomoses,[10] direct injection of esophageal veins[17] and perhaps hepatic artery ligation,[9] there is now a possibility of preventing recurrent hemorrhage. Thus, methods for accurately diagnosing esophageal varices are becoming of increasing importance.

The mortality in the operative treatment of esophageal varices due to cirrhosis has been high, and operation is only justifiable in view of the extremely high mortality without operation. The natural tendency is for the surgeon to desire to obtain his patients earlier in the disease before exsanguinating hemorrhage has occurred, and before liver function is greatly altered. Such prophylactic surgery could only be justified if the natural history of esophageal varices is well established, and surgery offers substantially longer survival.

For the purpose of establishing the relation of Laennec's cirrhosis to esophageal varices, 502 patients have been examined at the Veterans Administration Hospital at Hines, Illinois, over the past four years. The results of this study have been reported from time to time elsewhere. Because this is a continuing study, statistics extracted from our files at any one time will differ slightly from those on another date. In the course of this study valuable experience has been obtained in diagnostic techniques, and a good opportunity for the controlled evaluation of diagnostic procedures offered.

We present here the preliminary results of this study with special reference to the roentgenological examination techniques developed by one of us (I.E.K.), roentgen diagnostic criteria, and the comparison with esophagoscopic findings.

MATERIAL

All patients having hepatomegaly, splenomegaly or both, who have been admitted to the Veterans Administration Hospital,

* This paper represents one of a series of studies of disease of the liver and spleen initiated by Dr. L. A. Baker, Chief of Internal Medicine, Veterans Administration Hospital, Hines, Illinois.

† Physician-in-Charge, Diagnostic X-ray Service, Veterans Administration Hospital, Hines, Illinois.

‡ Section Chief in General Medicine, Veterans Administration Hospital, Hines, Illinois.

§ Assistant Chief of Medical Service, Veterans Administration Hospital, Hines, Illinois.

TABLE I

Cirrhosis—419 cases		Non-Cirrhotic—83 Cases	
Laennec's	370	Fat infiltration (early Laennec's)	56
Cause undetermined	28	Hepatomegaly, cause undetermined	13
Biliary cirrhosis	8	Hepatosplenomegaly, cause undetermined	4
Postnecrotic cirrhosis	7	Chronic hepatitis	4
Cardiac cirrhosis	5	Granuloma of the liver	2
Hemochromatosis	1	Portal hypertension with varices, cause undetermined	1
		Diabetes with hepatomegaly	1
		Inferior vena caval thrombosis	1
		Splenomegaly, cause undetermined, with varices	1

Hines, Illinois, have been studied in the past four years by a special board consisting of two or more internists and two or more vascular surgeons. In addition, all patients with liver dysfunction without hepatomegaly were likewise surveyed, provided a definite diagnosis of hepatitis or other known systemic disease causing liver dysfunction could not be established. In all, 502 patients have been thus studied at the time of writing. Table 1 demonstrates the distribution of this group clinically.

The routine survey on each patient consists of a complete history and physical examination, liver function tests including thymol turbidity, cephalin-cholesterol flocculation, serum alkaline phosphatase, total proteins, albumin-globulin ratio, bromsulphalein retention, cholesterol and cholesterol-ester ratios, quantitative Van den Bergh, prothrombin time, and additional tests as indicated. In addition to this, each patient was subjected to roentgen examination of the esophagus for varices and to esophagoscopy. Liver biopsy was performed in selected cases, when the diagnosis was in doubt. On each return visit, re-examination was carried out in all spheres including esophagoscopy, and roentgen examination as indicated.

ESOPHAGOSCOPY

For the purposes of this report the findings of the esophagoscopist were considered correct. Although such a procedure is essential in order to establish a baseline for comparison, it is not necessarily true. There is thus introduced an error of undetermined magnitude which tends to make roentgen examination of the esophagus appear less satisfactory than it truly is.

We know, for example, of a patient whose roentgenograms were reported positive for varices, and were reported by the resident who esophagoscoped him to show no varices. Recheck esophagoscopy within two weeks by an attending physician showed varices, confirming the roenrgenographic impression. Similarly, a patient who | showed no varices roentgenologically was reported to show "large varices" on esophagoscopy, but repeat esophagoscopy by an attending physician a short time later showed that these were large mucosal folds, and not varices.

The difficulties of esophageal examination by way of the esophagoscope are well known.[7] The uncooperative patient makes for a hasty view during which varices may be missed if indeed esophagoscopy can be accomplished at all. Esophageal folds may be mistaken for varices by the less experienced esophagoscopist and even at times by the most experienced. Hemangiomas[7] which are reported to cause confusion were not known to be encountered in our series.

Schindler[14] states that "esophagoscopy is assumed to have a morbidity rate of 2% and a mortality rate of 0.5%." We have had no mortality in over 1,000 esophagoscopies performed in this study. There has been the following morbidity. Hemorrhage followed esophagoscopy in 2 patients with esophageal varices, but neither pa-

tient died. Esophageal perforation occurred in 3 patients. All 3 patients recovered without subsequent sequelae other than a pulsion type diverticulum in 1 patient. Besides the above we have regularly noted pharyngeal pain as a benign but annoying sequela. This then gives us a mortality rate of 0 per cent and a morbidity rate of less than 1 per cent. No sequelae were noted from roentgenologic procedures. Esophagoscopy could not be accomplished due to lack of cooperation in 8 per cent of our patients, and was refused in an additional 10 per cent. These cases are, of course, not included in this report. No patients refused roentgen study. No laryngeal paralysis, as mentioned by Jackson and Jackson,[7] was encountered.

This is a clinical report, and autopsy studies are not included. The number of deaths to date has been too few for good analysis, and unfortunately routine injection studies have not been carried out. Thirty-two of 400 patients without varices have died and 30 of 102 patients with varices. It has been our experience that, as Boyd[4] points out, "They [varicosities] are easily overlooked at autopsy, for the varicosities collapse after death and may become invisible beneath the thick squamous epithelium of the esophagus."

ROENTGEN TECHNIQUE

Contrast Medium. When Wolf,[18] in 1928 first reported that esophageal varices could be shown on roentgenograms, he thought that a thin mixture of barium sulfate should be used. He found that with thin mixtures, even after complete emptying of the esophagus, one could see a delicate coating of barium in the crevices between the varices. His objection to thick mixtures was that they might "squeeze out" the blood from the varices by compressing them against the wall of the esophagus. Schatzki[13] used a contrast medium consisting of equal parts of barium sulfate and water. Hare *et al.*[5] used both thin and thick mixtures. We[8] have used in our examinations of the small intestine, a stable suspension of barium sulfate in water, made by adding a small amount of sodium carboxymethylcellulose. This substance, which is physiologically inert, as was learned by Necheles, Kroll, Bralow and Spellberg,[12] adds viscosity to the medium, and hence helps it cling to the mucous membrane. For routine roentgenograms of the esophagus, in which we aim to obtain a relief picture, the "thick medium" is used. This consists of 4 ounces of barium sulfate in 2 ounces of water, with 0.25 per cent carboxymethylcellulose added. For roentgenoscopy and spot roentgenograms of the filled esophagus, we use "regular medium," which is the same as that used for examination of the stomach and small intestine. This consists of 4 ounces of barium sulfate in 8 ounces of water, with 0.25 per cent carboxymethylcellulose added.

Position. The recumbent position was found to be better than the upright by Schatzki[13] who explained that in this position some opaque material might be forced back into the esophagus from the stomach. Hare *et al.*[5] advocate roentgenography in the upright and horizontal positions. We made roentgenograms in the upright position in some early cases, but, finding no advantage in it, settled on taking films in the recumbent position as a routine. The anteroposterior position is recognized by all to be practically worthless, because of the interference of the shadow of the spine. The case for roentgenography in both right anterior oblique and left anterior oblique positions was stated with imagination by Schatzki, who said that varices might "undulate in one plane only," so that films made in two positions at right angles to each other would be more likely to demonstrate the condition.

Phase of Breathing. Higgins[6] said that varices could best be brought out by having the patient "bear down" as in the Valsalva maneuver, and Benhamou *et al.*[1] also advocated this procedure. In Schatzki's experience, there was no particular merit in this maneuver, but he found there was some advantage in taking films in slight

inspiration, in order to avoid reading as varices the tortuous folds produced in the esophagus slackened by expiration. Our practice is to take three roentgenograms—an inspiratory and an expiratory in the right anterior oblique, and one in the left anterior oblique with the Valsalva maneuver.

Timing of Roentgenograms. When one uses "thin mixtures" of barium, the films may be quite unsatisfactory unless taken at precisely the right interval after swallowing. Our sodium carboxymethyl cellulose-barium suspension has the advantage of being quite viscous so that it clings to the mucous membrane of the esophagus for a time, and thus gives a mucosal relief picture. In some cases there is considerable latitude in the time for roentgenography after swallowing. For instance, in one case, films showed satisfactory mucosal relief at ten, fifteen and twenty seconds. In other cases, it is necessary, for the best results, to time the barium swallow carefully. We use a stop watch, and note in the roentgenoscopic room the interval from the command "swallow" to the time the bolus passes into the cardia. The technician uses the same medium and time for his roentgenograms. We have seen that in different patients, the transit time varies from five to thirty seconds, and exceptionally it may exceed one minute.

Roentgenoscopy. Only large varices are reliably seen roentgenoscopically. In the roentgenoscopic room we obtain spot films and note the transit time, but do not attempt always to decide as to presence or absence of varices. The spot films are made in the right anterior oblique and left anterior oblique positions, preferably with the patient recumbent.

Routine Procedure. In the roentgenoscopic room, spot roentgenograms are made in the right anterior oblique and left anterior oblique positions. A pair of exposures are made in each oblique—one with the esophagus filled with "regular medium," the other immediately after the medium has entered the stomach. Then, with the patient recumbent, a spoonful of the "thick medium" is placed in his mouth and, with a stop watch, the transit time is noted from the signal "swallow" to the appearance of the bolus in the stomach. This time is written on the back of the x-ray request, and the patient sent to the roentgenographic room. Here, the "thick medium" is used. One spoonful is given the patient before each film. The technician uses a stop watch to take films at the prescribed interval after the swallow. Three 14 by 17 inch film are made in the recumbent position. Two of these are in the right anterior oblique (one in the inspiratory phase, one in the expiratory phase). The third film made in the Valsalva maneuver is in the left anterior oblique position.

Technical Factors for Roentgenograms. Our films are made with these factors (in a patient of average size): kv. (peak), 116; ma., 200; time, 1/10 second. Added tube filtration, 2 mm. of aluminum.

Radiation Received by the Patient. A total of 3.6 r was measured on the skin of the patient (entrance skin dose) after the examination, including three roentgenograms, two spot-film exposures, and the incidental roentgenoscopy. It can be seen that reexamination for confirmation of a doubtful diagnosis subjects the patient to no serious radiation dosage.

COMPARISON OF ROENTGENOGRAPHIC AND ESOPHAGOSCOPIC DIAGNOSIS OF VARICES

We wish to be clear as to our method of tabulating our cases (Table II). The original roentgen report is the one tabulated, not the impression on reviewing the films. All cases are considered roentgenologically positive in which the conclusion of the roentgen report reads "evidence of varices," "probable varices," or "suggestive of varices."

It appears that the clinically important cases of varices were, in the main, diagnosed roentgenographically in that hemorrhage occurred in 31 of 66 cases in Group I (positive by both methods), but only in 1

Table II

	Group		No. of Cases
Agreement	I	Roentgen findings positive, esophagoscopy positive	66
	II	Roentgen findings negative, esophagoscopy negative	388
		Total cases with agreement (90% of all cases)	454
Disagreement	III	Roentgen findings negative, esophagoscopy positive	36
	IV	Roentgen findings positive, esophagoscopy negative	10
		Total cases with disagreement	46
Doubtful	V	Esophagoscopy suggestive, roentgen findings negative	2
		Total number of cases	502

There were 102 cases shown to have varices by esophagoscopy, representing 20 per cent of the entire group. Of these, 66 per cent were positive roentgenologically.

case in Group III (negative on roentgen ray, positive on esophagoscopy).

DISCUSSION

Our experience strongly impressed on us the necessity of a multiple film procedure. Because the esophageal walls retain their flexibility when varices are present, peristalsis may cause the varices to be ironed out at times. We found that frequently only two films, or even one, showed varices. Figures 1 and 2 show a proved case of varices in which, of two films made in the same position on the same day, one was

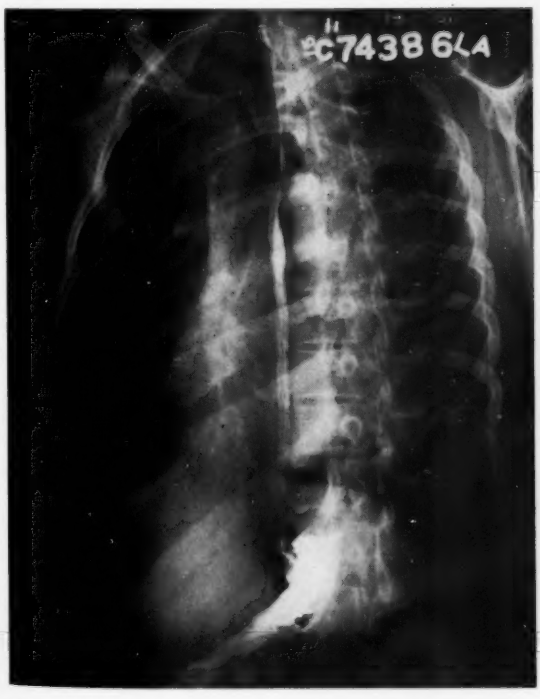

Fig. 1. Showing importance of multiple-film procedure. In this film, there is a good mucosal relief picture, with no evidence of varices.

Fig. 2. Same patient as in Figure 2, same day, same position. Extensive varices present, evidenced by scalloped borders, and thickened folds with rounded expansions.

TABLE III

TECHNICAL DEFECTS IN ROENTGENOGRAPHIC EXAMINATION

	Cases
A. Film made too soon after swallowing (no barium in lower esophagus)	13
B. Film made too long after swallowing (no mucosal relief)	3
C. Too few satisfactory films	8

positive for varices, the other negative. The examination not infrequently had to be repeated because of errors in technique. Because early varices were often missed by roentgenoscopy, it was not possible to order roentgenographic positions according to the roentgenoscopic observation, as is suggested by some writers.

The roentgenological examinations were made and reported by a number of members of the Roentgen Department, including residents, with varying or no consultation. They are tabulated in accordance with the actual report, even though, on review, they might be diagnosed differently. The radiologist reviewing the films (I.E.K.) had the advantage of knowing the esophagoscopic report. (It may be commented here that the endoscopist had an important initial advantage in this comparison, because roentgen examination was always made before esophagoscopy, and the results were made available before the esophagoscope was inserted.) On review of the films in Group III (roentgen findings negative, esophagoscopy positive), 8 of 36 cases are roentgen-ray positive. In Group IV (roentgen findings positive, esophagoscopy negative) 4 of 10 cases are negative on review.

The accuracy of roentgen diagnosis would appear in an even more favorable light if the technically inadequate examinations were eliminated from consideration. Thus, in Group IV (roentgen findings positive, esophagoscopy negative) 1 of 10 cases had a technically inadequate roentgen examination. In Group III (roentgen findings negative, esophagoscopy positive), 16 of 36 cases had technically inadequate films. Without these cases, there would be only 29 cases of disagreement, instead of 46.

These were the reasons for unsatisfactory films (some qualified under more than one heading) (Table III). Those not having at least two satisfactory ordinary roentgenograms and one spot film were placed in Class c. Unsatisfactory films can be classified as underexposed films, those made in the anteroposterior position or insufficiently oblique, and those showing a blurred outline of the esophagus due to too long an exposure time, so that cardiac motion was transmitted to the esophagus. It is stressed that improvements in our technical procedure were made throughout our study, so that, as a rule, the latest roentgenograms were the best technically.

The accuracy of the roentgen diagnosis

TABLE IV

(Compare with Table II)

Group			No. of Cases
Agreement	I	Roentgen findings positive, esophagoscopy positive	74 (66, +8 from III)
	II	Roentgen findings negative, esophagoscopy negative	392 (388, +4 from IV)
		Total cases with agreement (96% of all cases)	466
Disagreement	III	Roentgen findings negative, esophagoscopy positive	12 (8 to I, 16 eliminated)
	IV	Roentgen findings positive, esophagoscopy negative	5 (4 to II, 1 eliminated)
Doubtful	V		2
		Total number of cases	485

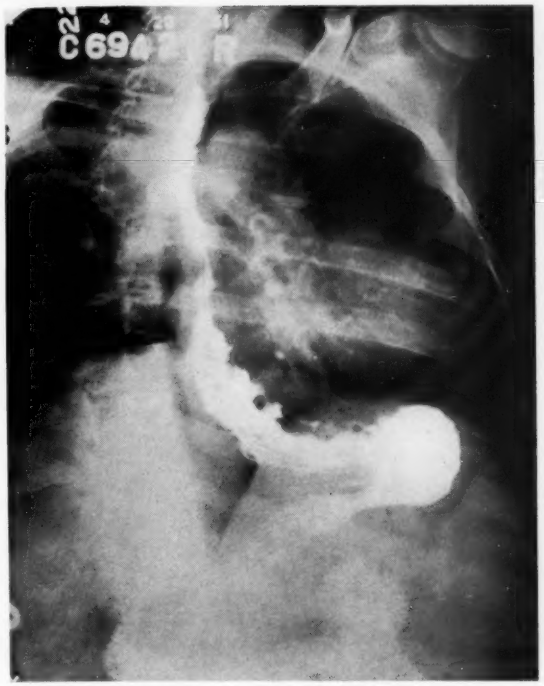

Fig. 3. Large varices, causing broadening and partial obstruction of lower esophagus, simulating hiatus hernia.

Fig. 4. Early varices. Scalloping of posterior border of filled esophagus.

in esophageal varices, it seems certain, can be increased to the extent that attention is paid to detail in technique, and diagnostic criteria thoughtfully applied.

These are the corrected figures (Table IV), after eliminating those cases with inadequate examinations, and correcting Table II to show changes of diagnosis on review of films. It is emphasized that the figures in Table IV represent a possibly attainable rather than an actual accuracy. They are shown in order to provoke thought and stimulate effort toward an ideal.

DIFFERENTIAL DIAGNOSIS

Air bubbles are excluded by their characteristic faceted appearance. "Curling" of the esophagus is distinguished from varices by the mucosal relief study and by the depth of the indentations. When extensive esophageal varices were present, a hiatus hernia was sometimes simulated (Fig. 3). This roentgenogram also shows widening and partial obstruction of the lower esophagus, which may be present in some cases. Benign tumors and carcinoma of the esophagus show a rigidity which varices lack, and should not be difficult to differentiate from them. In peptic esophagitis there is atypical scalloping of the

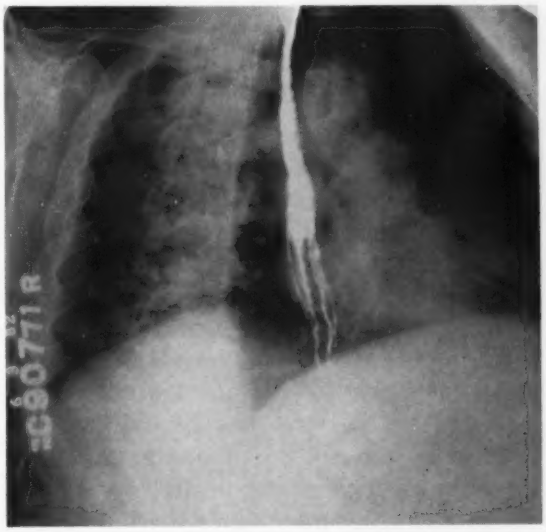

Fig. 5. Both positive signs of varices present.

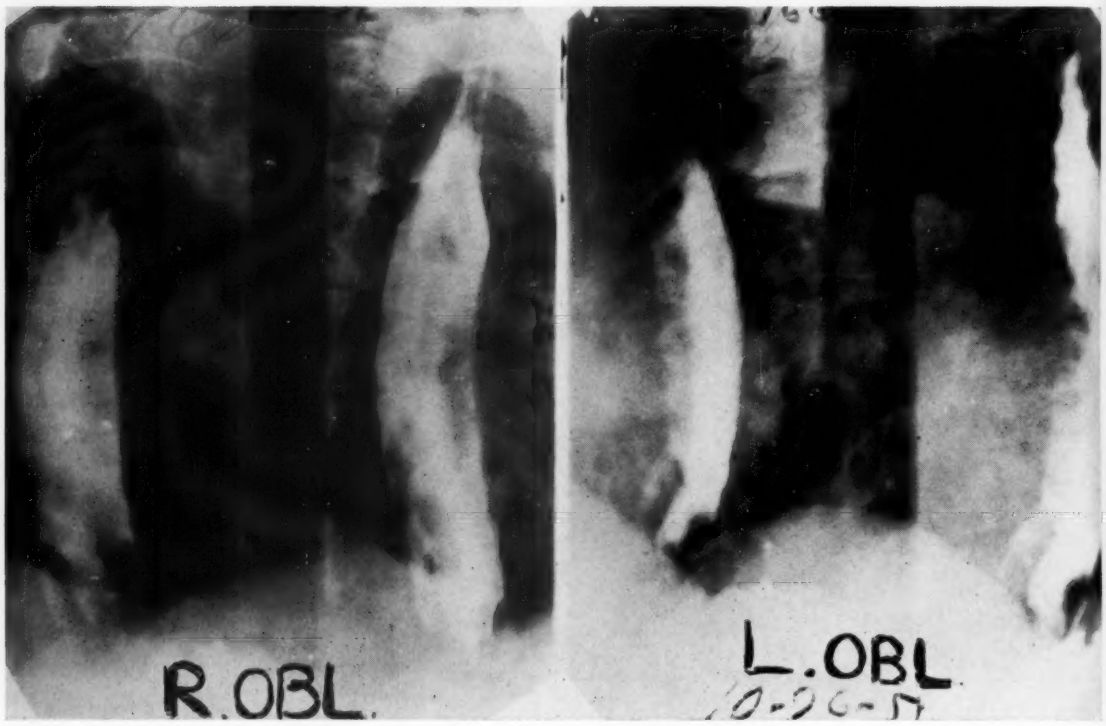

FIG. 6. Varices present. Both positive signs.

borders, having the appearance of thickened mucosal folds, rather than varices.

ROENTGENOLOGIC DIAGNOSIS

Large esophageal varices can be seen roentgenoscopically and by usual roentgenographic procedures. Extensive varices may also produce delay in emptying of the widened lower esophagus (Fig. 3). However, we sought dependable roentgenographic signs of smaller varices, which may be seen esophagoscopically, but have in the past been missed by routine roentgenography. We learned that thickened longitudinal folds did not mean varices. But the presence of thickened folds with rounded expansions seen on the mucosal relief films is an important sign of varices, and quite small ones may be demonstrated in this way. These often have a beaded appearance, but are to be distinguished from air bubbles. Scalloping of the borders of the filled esophagus (Fig. 4, 5, and 6) is a valuable sign, especially when the width of the defects varies between 0.5 and 1.0 cm., as it did in 87 per cent of the cases in which it was found. The combination of these two signs—characteristic scalloping, and thickened folds with rounded expansions (Fig. 5 and 6)—is strong evidence for varices.

CONCLUSIONS

1. In a study of 502 patients with diseases of the liver and spleen, the esophagus was examined for varices, in each case by both the roentgen ray and the esophagoscope. In the entire group, these two methods agreed 90 per cent of the time. In the group of 102 cases which were positive on esophagoscopy, 66 were positive on roentgen examination.

2. A standard procedure for roentgen examination is described, using as contrast medium a suspension in water of barium sulfate and sodium carboxymethylcellulose.

3. The early roentgenographic signs of esophageal varices are described. They are:

(a) characteristic scalloping of the borders of the filled esophagus, and (b) rounded expansions in the mucosal folds of the lower esophagus, seen on relief study.

I. E. Kirsh, M.D.
Veterans Administration Hospital
Hines, Illinois

REFERENCES

1. Benhamou, E., Damazer and Gardel. Etude radiologique des varices oesophagiennes. *J. de radiol. et d'électrol.*, 1950, *31*, 205.
2. Bennett, H. D., Baker, L., and Baker, L. A. Complications in use of esophageal compression balloons (Sengstaken tube). *A.M.A. Arch. Int. Med.*, 1952, *90*, 196–200.
3. Bockus, H. L. Gastroenterology. Vol. III. W. B. Saunders Co., Philadelphia, 1949.
4. Boyd, William. The Pathology of Internal Diseases. Lea & Febiger, Philadelphia, 1940.
5. Hare, H. F., Silveus, Esther, and Ruoff, F. A. Esophageal and gastric varices, with report of case. *S. Clin. North America*, 1948, *28*, 729–732.
6. Higgins, W. H., Jr. Esophageal varix; report of 115 cases. *Am. J. M. Sc.*, 1947, *214*, 436–441.
7. Jackson, C., and Jackson, C. L. Bronchoesophagology. W. B. Saunders Co., Philadelphia, 1950, p. 233.
8. Kirsh, I. E., and Spellberg, M. A. Examination of small intestine with barium-carboxymethyl cellulose suspension. *Radiology*. To be published.
9. Leger, L., Albot, G., Oudot, J., and Zerolo, J. La ligature de l'artère hépatique commune dans le traitement des cirrhoses. *Presse méd.*, 1952, *60*, 173–178.
10. Linton, R. R. Surgical treatment of bleeding esophageal varices by portal systemic venous shunts; with report of 34 cases. *Ann. Int. Med.*, 1949, *31*, 794–804.
11. McIndoe, A. H. Vascular lesions of portal cirrhosis. *Arch. Path. & Lab. Med.*, 1928, *5*, 23–42.
12. Necheles, H., Kroll, H., Bralow, S. P., and Spellberg, M. Peptic ulcer in man; new antacid made to meet requirements of antacid therapy, chemical and laboratory work. *Am. J. Digest. Dis.*, 1951, *18*, 1–7.
13. Schatzki, R. Roentgen demonstration of esophageal varices. *Arch. Surg.*, 1940, *41*, 1084–1100.
14. Schindler, R. Safe diagnostic optical esophagoscope. *J.A.M.A.*, 1948, *138*, 885–887.
15. Seabrook, D. B. Resection of lower esophagus for bleeding varices. *Northwest Med.*, 1948, *47*, 498–500.
16. Sengstaken, R. W., and Blakemore, A. H. Balloon tamponage for control of hemorrhage from esophageal varices. *Ann. Surg.*, 1950, *131*, 781–789.
17. Tolan, J. F. Esophageal varices and their treatment by injection. *Laryngoscope*, 1949, *59*, 425–431.
18. Wolf, G. Die Erkennung von Oesophagus-Varizen im Röntgenbilde. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *37*, 890–893.

GAS IN THE PORTAL VEINS OF THE LIVER IN INFANTS

A ROENTGENOGRAPHIC DEMONSTRATION WITH POSTMORTEM ANATOMICAL CORRELATION

By JOHN N. WOLFE, M.D., *and* WILLIAM A. EVANS, M.D.
From the Department of Radiology of the Children's Hospital of Michigan
DETROIT, MICHIGAN

IN FOUR infants a vascular pattern of gas in the liver has been observed in roentgenograms of the abdomen made fifteen hours, seven days, six hours, and fourteen hours prior to death. Postmortem observations have indicated that this gas is in the portal venous system. Two additional cases have presented similar postmortem findings without prior roentgen studies and these have been included in this report. It is the purpose of this communication to report this roentgen sign and its significance.

The material for this report is from the Children's Hospital of Michigan in the period from January, 1950, to February 5, 1954. The 6 cases studied were all infants who died and had postmortem examinations within this period.

A review of the literature published on the subject of liver, portal venous system and mesenteric veins for the ten year period from 1942 to 1952 fails to reveal any reference to this condition, and it is the opinion of the authors that this is an original observation.

The condition demonstrated by roentgenograms of the abdomen is quite characteristic. There are linear areas of radiolucency traversing the liver, appearing to gather or originate in the region of the hilus and then to traverse the liver in more or less horizontal fashion approaching the right lateral abdominal and chest walls. At first glance one may believe these to be bronchovascular radicles in the lung extending into the posterior costophrenic sinus behind the liver. However, the general pattern of lung markings in the more dependent portions of the lower lobes is in a more cephalocaudad direction whereas the markings in question are more transverse and tend to converge upon the hilus of the liver. It was not at first evident from the roentgen appearance of the gas pattern whether the gas was in the biliary radicles or in the circulatory system and the possibility of a cholangitis with gas forming organisms was entertained. Postmortem observations have indicated that the gas is in the portal venous system.

The following are the reports of the cases observed.

REPORT OF CASES

CASE 1 R.A. (CH 51640), white male, aged seventeen days at death. The interval between death and autopsy, six hours and fifteen minutes.

Clinical Diagnosis. (1) Intestinal obstruction, mechanical with peritonitis.

Anatomical Diagnosis. (1) Pneumatosis intestinalis, jejunum, ileum and colon; (2) gas thrombi, extensive, in the mesenteric veins, vena cava and right atrium; (3) postoperative functioning colostomy with imperforate anus and vesicorectal fistula; (4) pneumonia.

Present Illness and Hospital Course. The infant was first seen in this hospital at the age of two hours for an imperforate anus with a rectovesical fistula. A colostomy was performed and the patient was discharged ten days later in good condition. Thirty-six hours after the patient arrived home, projectile vomiting was observed by the parents together with profuse, frequent, watery stools from the colostomy. He was then re-admitted and general supportive measures were undertaken including oxygen therapy, penicillin, streptomycin, sulfadiazine and intravenous fluids. He continued acidotic and died nineteen hours after admission. A roentgen examination of the abdomen made fifteen hours prior to death revealed a pattern of gas in the liver which was thought to be within the circulatory system or in the biliary radicles of the liver.

The significant findings at postmortem examination included the presence of gas in the thin-walled mesenteric veins together with some blood. Gas was also present in the right atrium and inferior vena cava. There was a rather

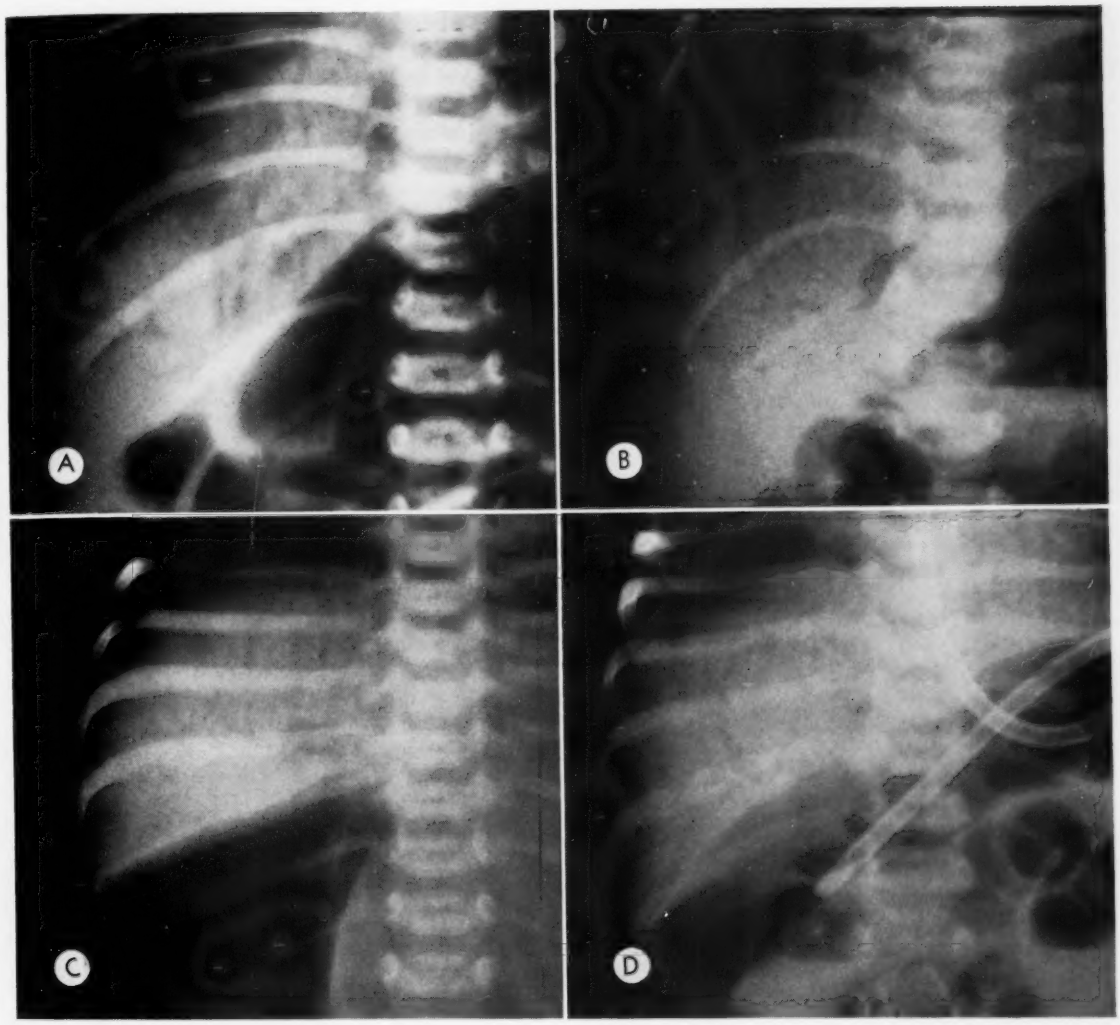

Fig. 1. Portions of abdominal roentgenograms exposed in the anteroposterior projection showing patterns of gas in the portal veins of the liver. (*A*) and (*B*) Case I. (*C*) Case III. (*D*) Case II.

marked amount of submucosal and subserosal air throughout the intestine from the jejunum through the colon. The liver weighed 175 gm. as compared to the normal 123 gm. There was no evidence of gas within the gallbladder or common bile duct. On microscopic examination of the liver, the usual hepatic structures were present. The cells were noted to be somewhat cloudy and granular. Postmortem blood culture revealed the presence of *E. coli* and antemortem blood culture was negative.

Case II. R.B. (CH 68853), white male, aged thirty-five days at death. The interval between death and autopsy, fifteen minutes.

Clinical Diagnosis. (1) Duodenal atresia, postoperative; fetal peritonitis.

Anatomical Diagnosis. (1) Intestinal obstruction, mechanical, jejunal secondary to duodenojejunostomy; (2) peritonitis, extensive, chronic, fibrous; (3) pneumonia, extensive, right upper and middle lobes; (4) gas thrombi, extensive, in the mesenteric veins; (5) peritonitis, meconium; (6) necrotizing enteritis, small intestine.

Present Illness and Hospital Course. The infant presented the symptoms and signs of a high small intestinal obstruction as manifested by persistent and early vomiting of bile-stained material. He passed one meconium stool. The initial roentgenograms of the abdomen demonstrated a complete obstruction and evidence of meconium peritonitis. At operation on the second day of life an atresia was found in the distal duodenum, a 10 cm. length of small intestine was resected and a duodenojeju-

nostomy performed. The immediate postoperative period was uneventful. However, obstruction developed on the third postoperative day at the anastomotic site and the course then became retrogressive to his death. A roentgen examination of the abdomen, made seven days prior to death, revealed a vascular pattern of gas within the liver.

The significant findings at autopsy included a rather extensive peritonitis as indicated by fibrous adhesions between all loops of bowel and neighboring structures. An obstruction in the jejunum was demonstrated at the anastomotic site. The liver weighed 210 gm. (127 gm. normal) but did not appear otherwise remarkable. Unfortunately, no negative or positive statement was made regarding the mesenteric veins. On microscopic examination, severe necrotizing enteritis was evident. Nothing was seen to suggest a diagnosis of pneumotosis intestinalis. The postmortem blood culture revealed the presence of *E. coli*.

CASE III. D.N. (CH 87352), white male, aged fifteen days at death. Interval between death and autopsy, three hours, ten minutes.

Clinical Diagnosis. (1) Erythroblastosis fetalis; (2) septicemia; (3) peritonitis.

Anatomical Diagnosis. (1) Erythroblastosis fetalis; (2) submucosal emphysema of the ascending colon; (3) gas thrombi in the mesenteric veins; (4) vascular thrombosis, left occipital and right frontal lobes of the brain; (5) bronchopneumonia.

Present Illness and Hospital Course. This infant was first seen on the day of birth for erythroblastosis fetalis. He received an exchange transfusion and the course was uneventful except that he improved slowly and required oxygen therapy. He was discharged after seven days. Four days after arriving home and two days prior to the second and final admission and at fourteen days of age, he refused to eat and regurgitated any forced feeding. Respirations were loud and grunting. He was given an enema at home, and following this, there were numerous watery stools which, however, ceased the day of admission. The abdomen became distended and the infant appeared markedly dehydrated. He died seven hours after admission to the hospital.

A roentgen examination of the abdomen made six hours prior to death revealed a vascular pattern of gas in the liver.

The significant findings at autopsy were the presence of numerous gas bubbles within the portal vein and the absence of gas within the gallbladder which was opened under water. The entire bowel was hyperemic and distended with liquid feces. There were numerous emphysematous blebs in the submucosa of the ascending colon. Microscopically the supportive cellular infiltrations associated with pneumatosis intestinalis were not observed. No peritonitis was observed and the sections of the liver were not remarkable. Postmortem blood culture was positive for *E. coli*.

CASE IV. J.H. (CH 91990), white male, aged sixteen days at death. Interval between death and autopsy two and one-half hours.

Clinical Diagnosis. (1) Erythroblastosis fetalis; (2) diarrhea.

Anatomical Diagnosis. (1) Erythroblastosis fetalis; (2) diarrhea, clinical; (3) pulmonary edema and focal atelectasis and hemorrhage with emphysema and bronchopneumonia; (4) focal hepatic necrosis with inspissation of bile in cells and canaliculi; (5) gastroenteritis; (6) bilateral renal cortical necrosis.

Present Illness and Hospital Course. The infant was admitted at the age of one day for erythroblastosis fetalis. He was discharged at the end of five days after general supportive measures. He was not given transfusions. He was re-admitted five days later for a severe anemia and jaundice; the hemoglobin was 6 gm., otherwise the child was very well. During the second hospital admission, he was transfused repeatedly with packed red blood cells until the hemoglobin was 15.1 gm. per 100 cc. On the day of the second admission, the patient had two loose stools and two days later very definite diarrheic stools, and treatment was instituted for this condition. The patient was placed on penicillin and sulfadiazine and given intravenous fluids. Despite this, the diarrhea persisted and he became more dehydrated and died on the sixth day of the second hospital admission. The significant finding at autopsy was the observation of numerous variable sized gas bubbles within the portal venous system. No gas was found in the gallbladder or the biliary radicles. The liver weighed 170 gm. (123 gm. normal). There were small areas of focal necrosis as seen on the microscopic sections of the liver with inspissation of bile in some of the canaliculi and bile ducts. Postmortem blood culture revealed the presence of *E. coli*.

CASE V. E.B. (CH 98364), Negro female, aged three months at death. The interval between death and autopsy was eight hours and thirty-five minutes.

Clinical Diagnosis. (1) Diarrhea.

Anatomical Diagnosis. (1) Diarrhea (clinical) etiology unknown; (2) early serosal peritonitis; (3) pneumatosis intestinalis; (4) gas in portal veins and tributaries.

Present Illness and Hospital Course. The infant was first admitted to this hospital at nine weeks of age because of intermittent diarrhea for four weeks and vomiting for three days. The birth weight was 5 lb. 1 oz. and the weight on admission 7 lb. 12½ oz. Mild dehydration was observed and treatment included penicillin, sulfadiazine and intravenous fluids. She was discharged improved after five days weighing 8 lb. 11½ oz. However, she was re-admitted ten days later because of persistent diarrhea and vomiting, and weighing 8 lb. ½ oz. There was marked dehydration and treatment included intravenous fluids, adrenal cortical extract, oxygen and transfusions of plasma and blood. A marked abdominal distention was observed on the sixth hospital day and death occurred seven hours later. No roentgen examinations were made during the patient's illness.

The gross autopsy findings which seemed significant were a marked abdominal distention, 30 cc. of serosanguineous fluid in the peritoneal cavity, a pneumatosis on the antimesenteric surface of the duodenum and upper jejunum with small air bubbles in the mesentery and in the iliac arteries. The vessels of the liver were ligated and a roentgen examination of the liver revealed a pattern of gas in the portal venous system. Microscopic findings included congestion of the lungs, spleen and liver with some striations of the liver, extensive destruction of the intestinal mucosa, and "air cells" in the submucosa of the colon. A heavy growth of *E. coli* was obtained by blood and lung culture.

Case VI. D.B. (CH 105798), white male, aged nineteen days at death. Interval between death and autopsy ten hours ten minutes.

Clinical Diagnosis. (1) Pneumatosis intestinalis; (2) erythroblastosis fetalis.

Anatomical Diagnosis. (1) Diarrhea (clinical); (2) acute gastroenteritis; (3) gas in the portal veins; (4) aspiration pneumonia.

Present Illness and Hospital Course. This infant was first admitted to the hospital on the first day of life because of severe jaundice. The liver and spleen were palpable. The Coomb's test was positive and a diagnosis of erythroblastosis fetalis type A c DE was made. Treatment consisted of cortisone, adrenocorticotropic hormone, procaine penicillin, sulfadiazine, and synkamen, and he was discharged after four days. At eleven days he was re-admitted for transfusion of 50 cc. of blood. The stools had been loose for two days. The third and final admission occurred at eighteen days of age. The stools had been soft and frequent but not watery for two days. There had been no vomiting but all feedings were refused the day of admission. The baby was blue-gray in color with deep puffing respirations, a depressed fontanelle, and a distended tympanitic abdomen. A roentgen examination fourteen hours before death revealed a gaseous and liquid distention of the bowel with a vascular pattern of gas in the liver. Death occurred in spite of the usual supportive measures.

The significant gross postmortem findings were a marked abdominal distention, mucosal congestion and ecchymosis of the small intestine and colon without ulceration. Fine gas bubbles were detected in the veins of the mesentery of the small intestine and in the portal vein. Microscopic studies yielded no additional information of significance. Postmortem culture of the heart blood produced a heavy growth of *E. coli.*

COMMENT

These 6 cases exhibiting gas in the portal veins of the liver have in common the features of being infants who died with marked gaseous distention of the bowel and postmortem blood cultures, yielding a growth of *E. coli.* Thus, it is reasonable to suppose that gas under increased tension in the lumen of the bowel enters the mesenteric veins and collects in the portal system of the liver. This concept is supported by the demonstration of pneumatosis of the bowel in 3 cases.

This phenomenon seems to be not directly related to the primary disease which was erythroblastosis fetalis in 3 cases, duodenal atresia in 1 case, imperforate anus Type III with rectovesical fistula in 1 case, and infantile diarrhea, 1 case. Death occurred at fifteen, sixteen, eighteen, nineteen and thirty-five days, and three months of age following a terminal episode of vomiting or diarrhea with abdominal distention. Inflammatory changes were not demonstrated in the liver or biliary system in any of the cases by gross or microscopic postmortem examination.

William A. Evans, M.D.
552 Fisher Building
Detroit 2, Michigan

POSTCAVAL URETER

A CASE REPORT

By D. A. VAN VELZER, M.D., C. W. BARRICK, M.D., *and* E. L. JENKINSON, M.D.
Department of Radiology, St. Luke's Hospital
CHICAGO, ILLINOIS

THIS is a case presentation of a postcaval ureter, which was diagnosed preoperatively.

CASE REPORT

This patient, J.C., an obese white male, aged fifty-three, was admitted to St. Luke's Hospital on May 14, 1954, because of right lower quadrant pain and abdominal distention.

The patient states that for the last two years he has had malaise and dull abdominal pain. About one month previous to this admission, the patient began to have nocturia, one to two times nightly. One night he developed a severe right lower quadrant pain which radiated to the midline below the umbilicus. He had an urgent desire to void, but the amount of urine produced was minimal. Micturition aggravated the above pain and produced a sharp pain in the penis. He developed shaking chills and fever of 102° F. His abdomen became distended, and after attempting to void, he found that he could not zip up his trousers. He had abdominal discomfort at stool, and any jarring motion aggravated the abdominal pain.

The patient was admitted to St. Luke's Hospital following the above episode, with an initial diagnosis of acute appendicitis. Physical examination on admission revealed a temperature of 102.2° F., orally, right lower quadrant guarding and rebound tenderness, and hyperactive bowel sounds. The rectal examination was normal and there was no costovertebral angle tenderness present. The initial urinalysis revealed one to two epithelial cells and two to three leukocytes per high power field. The white blood cell count was 9,700. There were 13.8 grams of hemoglobin present and 4.92 million red blood cells. Three hours after admission the white blood cell count rose to 13,000, with 84 per cent polymorphonuclears, 3 per cent band forms, and 13 per cent lymphocytes. The patient was treated conservatively with penicillin, hot foments, and surgical liquids. The day after admission, the white blood cell count dropped to 7,900, with normal differential. It remained within normal limits thenceforth. The abdominal distention gradually improved and the patient felt moderately well at the end of his five day hospitalization.

FIG. 1. Preliminary film with retrograde catheter in place. There is residual contrast material in the colon. The course of the catheter passes just medial to the pedicle of the fourth lumbar centrum (see Fig. 3).

During the next month, the patient's abdominal symptoms persisted moderately, and he was re-admitted to St. Luke's Hospital on June 8, 1954. The white blood cell count and urinalysis on this admission were normal except for a few bacteria in the urine. The erythrocyte sedimentation rate was 23 mm. per hour on admission by the Westergren method. The physical examination at this time revealed no change from the physical examination at the time of the previous admission. An intravenous urogram showed delay in appearance of the contrast material at five minutes in both kidneys, the right ureter not being visualized. Subsequent films up to forty-five minutes showed pyelectasis and ureterectasis on the right. The right ureter extended to the level of

the fourth lumbar centrum, where it turned abruptly to the midline and rapidly narrowed in caliber. It was noted on one of the films to be of normal caliber in its pelvic portion where it was lying more medially than usual. No lesions were seen on the left. A diagnosis of retrocaval ureter was made. This was confirmed on subsequent retrograde pyelography, which showed the ureter to pass just medial to the right pedicle of L-4. Surgical exploration on June 17, 1954, confirmed the roentgenographic diagnosis. The ureter was transected and an end-to-end anastomosis was performed after the retrocaval portion was dissected out. Postoperative recovery was uneventful. (Surgeon, T. P. Grauer, M.D.)

Fig. 3. Urogram showing course of the ureter after withdrawal of the catheter. Lower right ureter lies more medial than usual and has a narrow caliber.

EMBRYOLOGY

The embryology of the anomalous condition has been described in detail by many authors (Graves and Davidoff,[3] Randall and Campbell,[8] Pick and Anson,[7] and others). The basis for the malformation is as follows: During fetal life, the metanephros (permanent kidney) ascends from the pelvis and passes through a venous anastomotic ring composed of three pairs of venous systems. The largest vein and first to appear is the postcardinal vein, which lies laterally. The second lies ventromedially and is the subcardinal vein. The third lies dorsomedially and is the supracardinal vein. These veins are symmetrical bilaterally and form an anastomotic ring surrounding the fetal aorta. The kidney passes within this ring, medial to the postcardinal vein in its ascent, and then passes laterally behind the postcardinal vein and its anastomotic branches with the supracardinal vein to its final position in the renal fossa. In later fetal life, the infrarenal part of the postcardinal vein atrophies, and the lumbar part of the inferior vena cava is formed by the fusion of the right and left supracardinal veins. Thus, the ureter will then lie lateral to the vena cava in its normal position. However, if the postcardinal vein persists in any manner, the ureter will certainly encircle it. There are several obvious anomalous possibilities, the most common of which is the persistence of the right postcardinal vein to form the inferior vena cava, in which case the ureter usually encircles the vena cava at about the level of the third lumbar vertebra, the ureter being compressed anteriorly by the vena cava which fails to atrophy.

Fig. 2. This shows the right renal pelvis and proximal ureter dilated above the level of obstruction.

The second possibility is a double vena cava on the right with the ureter passing through a ring formed by the veins and their anastomoses. The third possibility

reported is a double vena cava (one on each side) with the right ureter encircling the right one. There has been one reported case of double vena cava with each ureter encircling its corresponding vein.

The explanation for the fact that the right postcaval ureter is found more often than the left is simply that the right postcardinal vein persists longer in fetal life than the left.

SUMMARY

A case report and embryology of postcaval ureter diagnosed preoperatively is presented with the accompanying roentgenograms. There are less than one hundred cases in the literature.

E. L. Jenkinson, M.D.
St. Luke's Hospital
1439 South Michigan Ave.
Chicago 5, Illinois

REFERENCES

1. CATHRO, A. J. M. Section of inferior vena cava for retrocaval ureter; new method of treatment. *J. Urol.*, 1952, *67*, 464–475.
2. DERBES, V. J., and DIAL, W. A. Postcaval ureter. *J. Urol.*, 1936, *36*, 226–233.
3. GRAVES, R. C., and DAVIDOFF, L. M. Anomalous relationship of right ureter to inferior vena cava. *J. Urol.*, 1922, *8*, 75–78.
4. GRUENWALD, P., and SURKS, S. N. Pre-ureteric vena cava and its embryological explanation. *J. Urol.*, 1943, *49*, 195–201.
5. HESLIN, J. E., and MAMONAS, C. Retrocaval ureter; report of four cases and review of literature. *J. Urol.*, 1951, *65*, 212–222.
6. MULLEN, W. H., JR., and ENGEL, W. J. Circumcaval ureter. *Radiology*, 1952, *59*, 528–537.
7. PICK, J. W., and ANSON, B. J. Retrocaval ureter; report of case, with discussion of its clinical significance. *J. Urol.*, 1940, *43*, 672–685.
8. RANDALL, A., and CAMPBELL, E. W. Anomalous relationship of right ureter to vena cava. *J. Urol.*, 1935, *34*, 565–583.
9. SHIH, H. E. Postcaval ureter. *J. Urol.*, 1937, *38*, 61–66.

Vol. 74, No. 3

STUDIES ON NAGASAKI (JAPAN) CHILDREN EXPOSED *IN UTERO* TO THE ATOMIC BOMB*

A ROENTGENOGRAPHIC SURVEY OF THE SKELETAL SYSTEM

By WATARU W. SUTOW, M.D.,† *and* EMORY WEST, M.D.
From the Laboratories of the Atomic Bomb Casualty Commission

ANIMAL experiments have demonstrated the sensitivity of the developing embryonic and fetal skeletal system to radiation injury.[8,9,13,14,18,20] Skeletal anomalies of various types appear to be among the consistent and frequent abnormalities that follow intrauterine radiation, depending on the radiation dose and the stage of embryonic or fetal development at the time of exposure.

At the time of the atomic bomb explosions in Japan women in various stages of pregnancy were exposed to radiation at short distances. Thus, a certain number of children living at present in the atomic bombed cities were exposed during embryonic and fetal development. These children have been given comprehensive pediatric examinations. This report summarizes the results of the roentgenographic survey of the skeletal system to determine the occurrence of skeletal malformations in the Nagasaki children.

MATERIALS AND METHODS

Seventy-four children comprised the "exposed" group. This number represented the entire available group of children remaining in Nagasaki in 1952 who were known to have been *in utero* at the time of the atomic bomb explosion in Nagasaki and were within 2,000 meters of the hypocenter.*

Ninety-one children were, for the purposes of this study, considered as "controls." These children were also known to have been *in utero* on August 9, 1945. They were exposed to the atomic bomb, however, at distances of 4,000 to 5,000 meters from the hypocenter. At 4 to 5 kilometers, the biologic effects of atomic bomb radiation were considered to be negligible.

The method of selection of these two groups of children for investigation has been described elsewhere.[21] From a total of 1,771 female survivors of child-bearing age who were exposed to the bomb within the 2,000 meter area, 99 were found to have been pregnant at the time of exposure. Of the children resulting from these pregnancies, 75 were alive in 1952. A statistically comparable group of 1,744 women of child-bearing age were selected as controls from female survivors exposed at 4,000 to 5,000 meters. One hundred and thirteen of these women were pregnant on August 9, 1945. Of the children resulting from these pregnancies, 98 were alive in 1952. One child in the exposed group and seven children in the control group were not available for examination in 1952. Among the "exposed" group seen in 1952, major symptoms of radiation injury (epilation, purpura and/or oropharyngeal lesions) had occurred in 15 mothers.

The stage of gestation of any particular child at the time of exposure could not be accurately determined from existing records. The approximate gestational age at exposure in terms of "probable trimester of pregnancy," therefore, was calculated from the birth date of the children as follows:

Birth Date of Child	*Estimated Gestational Age at Exposure*
After Feb. 10, 1946	First trimester
Nov. 10, 1945 through Feb., 1946	Second trimester
Aug. 9, 1945 through Nov. 9, 1945	Third trimester

* Hypocenter was defined as the point on the ground directly over which the atomic bomb was calculated to have exploded at Nagasaki on August 9, 1945.

* This work was sponsored by the Atomic Bomb Casualty Commission, National Academy of Sciences-National Research Council, with funds supplied by the United States Atomic Energy Commission.

† Present address: M. D. Anderson Hospital, Houston, Texas.

TABLE I

CHILDREN EXPOSED *in utero* TO RADIATION FROM THE ATOMIC BOMB IN NAGASAKI AND EXAMINED IN 1952

Sex	Gestational Age at Exposure	Number Exposed within 2,000 Meters	Number Exposed at 4,000 to 5,000 Meters
Male	First trimester	13 (2)*	10
	Second trimester	10 (3)	14
	Third trimester	11 (2)	15
Female	First trimester	9 (4)	17
	Second trimester	14 (2)	18
	Third trimester	17 (2)	17
	Total	74	91

* Figures in parentheses indicate mothers with radiation symptoms.

The distribution of the children in terms of exposure status, sex and probable gestational age at exposure is summarized in Table 1.

In a 1951 study, roentgenograms obtained for the estimation of skeletal maturation included views of the left hand and wrist, left elbow, left shoulder, left knee and left foot and ankle. In 1952, lateral views of the skull, anteroposterior and lateral views of the entire vertebral column, and anteroposterior views of the lower legs were also taken. In all cases, therefore, it was possible to examine the entire left wrist and hand; the proximal one-third and distal one-third of the left radius and ulna; the distal one-third and proximal one-third of the left humerus; skull; rib cage; entire vertebral column; distal one-third of the left femur; entire tibia and fibula of both legs; left ankle and left foot. In most cases, the anteroposterior view of the dorsal and lumbar spines included the upper extremities as well, so that the humerus, radius and ulna of both sides were roentgenographed in their entirety; also the heads of both femurs were partially or wholly visible.

The lateral skull roentgenogram was examined to exclude abnormalities of the sella turcica and to note anomalies of shape, size and structure of the skull. In the examination of the vertebrae, a special effort was made to detect the failure of segmentation, variations in number, hemivertebrae, fusion of vertebrae, scoliosis, lumbosacral variations and spina bifida occulta. Rib abnormalities that were looked for included fusion of ribs, and variations in size and number.

In the hand and foot, the presence or absence of polydactylism, syndactylism and variations in number and appearance of ossification centers were noted. In particular, the occurrence of hypoplasia of the middle phalanx of the fifth finger and of accessory epiphyses at the bases of the metacarpals were investigated.

Failure of the laminae of the neural arch to fuse posteriorly was classed as "spina bifida occulta." The diagnosis of "lumbarization" was made when the first sacral segment had the appearance of a lumbar vertebra; that is, when it was not fused with the remaining sacral segments. The diagnosis of "sacralization" was made when one or both transverse processes of the vertebra were fused with the sacrum or articulated with the sacrum or ilium, leaving only four lumbar-type vertebrae. A rudimentary or a well-developed rib at the level of the seventh cervical body was called "cervical rib." "Lumbar ribs" included short ribs articulating with the lumbar vertebral bodies, usually the first lumbar. When the middle phalanx of the little finger, on inspection, appeared disproportionately short, the condition was classified as "hypoplasia." The short, normally contoured phalanx was not differentiated from the short deformed phalanx. "Accessory epiphyses" at the bases of metacarpal bones were noted when such epiphyses were either completely separated from, or only partially united with, the metacarpal shafts.

RESULTS AND DISCUSSION

1. *Neural Arch Defects.* In 38 of 72 (52.7 per cent) exposed and in 57 of 91

TABLE II

INCIDENCE OF NEURAL ARCH DEFECTS

		Number Examined	Proportion with Neural Arch Defects			
			G.A. I*	G.A. II	G.A. III	Total
Exposed	Boys	34	6(2)/13†	6(1)/10	5/11	17(3)/34
	Girls	38	4(1)/8	8(1)/14	9(3)/16	21(5)/38
	Total	72	10(3)/21	14(2)/24	14(3)/27	38(8)/72
Control	Boys	39	6(2)/10	12(4)/14	12(2)/15	30(8)/39
	Girls	52	9(2)/17	5(1)/18	13(3)/17	27(6)/52
	Total	91	15(4)/27	17(5)/32	25(5)/32	57(14)/91
Grand total		163	25(7)/48	31(7)/56	39(8)/59	95(22)/163

* G.A. refers to gestational age on August 9, 1945, I, II and III indicating first, second and third trimesters of intrauterine existence, respectively.

† Number in parentheses refers to children with failure of fusion of posterior laminae of the lumbar vertebrae, either alone or in combination with abnormality of sacral vertebrae.

(Note: In 2 children, roentgenograms of the vertebrae were not taken, thus reducing the total number of subjects to 163.)

(62.6 per cent) control children, the laminae of either the lumbar vertebrae (usually the fifth lumbar segment) or the sacral vertebrae or both failed to unite posteriorly. The over-all incidence was 58.3 per cent (Table II). The lumbar vertebrae were involved, either alone or in association with sacral vertebrae, in 13.5 per cent (22/163) of the children. The sacral vertebrae alone were affected in 44.8 per cent (73/163). There was no statistically significant difference in the incidence of these anomalies in relation to exposure to radiation, sex of children, or gestational age at exposure.

The incidence of "spina bifida occulta" found in the study was higher than has been observed in the literature available to us. Bucy,[2] after reviewing the literature, concluded that the probable incidence of spina bifida occulta in the general population approximated 5 per cent. Cushway and Maier[4] reported the somewhat higher incidence of 17 per cent among 931 adults. Giles,[5] in a study of lumbosacral vertebrae in 1,122 roentgenograms from "negative G-U examinations" in a consecutive series of 8,000 patients, reported spina bifida occulta in 267 cases, an incidence of 23.9 per cent. Brailsford[1] in a study of 3,000 roentgenograms of the spine found spina bifida occulta of the fifth lumbar vertebrae in 5 per cent and of the first and second sacral vertebrae in 11 per cent.

The extremely high number of neural arch defects found in this study immediately raised some question regarding the validity of calling this condition spina bifida occulta. It was possible that the failure of fusion simply represented a normal developmental process in which union will occur at later age periods. The skeletal development or rate of maturation in Japanese children, as assessed from hand and wrist roentgenograms, is twelve to twenty-four months slower than that of American children of comparable age and sex.[17] Similar retardation in the maturation of the vertebral components could explain the persistence of spina bifida occulta in older age groups.

Some American textbooks[3,10,11] indicate that the ossification of the vertebral neural arch is completed during the first two postnatal years. If this were also true of Japanese children, the failure of fusion in the children in this study, whose chronological ages range from six years and eight months to seven years and five months, represented unlikely degrees of developmental retardation in ossification. Certain

TABLE III

INCIDENCE OF ANOMALIES OF HAND BONES

		Number Examined	Proportion with Anomalies of Hand Bones			
			G.A. I	G.A. II	G.A. III	Total
Exposed	Boys	34	2/13	2/10	4/11	8/34
	Girls	39	3/9	4/14	2/16	9/39
	Total	73	5/22	6/24	6/27	17/73
Control	Boys	39	2/10	5/14	3/15	10/39
	Girls	52	3/17	8/18	10/17	21/52
	Total	91	5/27	13/32	13/32	31/91
Grand Total		164	10/49	19/56	19/59	48/164

investigators, however, have reported later age periods for the fusion of the laminae. Girdany and Golden[6] gave the age range of one to seven years for such fusion. Scammon[15] clearly indicated the ages seven to fifteen years for fusion of the laminae of the typical sacral vertebrae, but the time of fusion of the laminae of the lumbar vertebrae was not clearly defined.

Further investigations have been started to determine whether or not similar incidences of neural arch defects exist in older children and to derive, if possible, from such studies of other age groups more information regarding the course of developmental processes in the posterior bony arch of the lumbar and sacral vertebrae. Spina bifida occulta was not found outside the lumbosacral area.

2. *Anomalies of the Hand Bones.* The abnormalities most frequently found in roentgenograms of the left wrist and hand were hypoplasia of the middle phalanx of the fifth digit, accessory epiphyses at bases of the metacarpals (especially the second metacarpal) and an accessory secondary ossification center for one of the carpal bones. The incidence of these three types of anomalies has been summarized in Table III. No significant differences in the incidence of the anomalies were found between the boys and girls and between exposed and control groups.

The middle phalanx of the fifth digit has been found to be hypoplastic in a small number (1 per cent) of children.[3] This anomaly is prominent in mongolism[7] and also is found as a dominant hereditary characteristic.[19] The unexpectedly frequent occurrence of this particular characteristic among Japanese children has been noted by Pryde and is being investigated in detail.[12] In this series, the over-all incidence of the hypoplasia of the middle phalanx of the fifth digit was 42/164.

In the majority of cases, the hypoplasia of the phalanx was associated with distortion of the usual contour of the bone. In others, the bone appeared abnormally short without any change in its shape. Notching at the proximal end of the metacarpals, most frequently the second metacarpal, presumably represents an accessory ossification center.[3] This anomaly was found in 10 out of 164, or in 6.1 per cent of the children. Hypoplasia of the phalanx occurred in association with an accessory metacarpal epiphysis in 4 children, an incidence of 4/164.

Brailsford[1] differentiated between "pseudo-epiphysis" and "true supernumerary epiphysis." In all 10 children (10/164) in this series in whom this metacarpal abnormality was found, there was bony continuity between the diaphysis and the epiphyseal center. In a study of 1,000 "apparently normal children," Brailsford found 83 instances of pseudo-epiphyses of the metacarpal bones, 62 at the proximal portion of the second metacarpal and 21 at the distal end of the first metacarpal. Boden (quoted in Brailsford) in a study of diabetic children found pseudo-epiphysis of the second metacarpal in 14.7 per cent of boys and 5.3 per cent of girls.

An accessory epiphysis of the distal end of the first metacarpal was found in 1 child (exposed, third trimester girl), an accessory lunate in 1 (control, second trimester girl), an accessory lunate and greater multangular in 1 (control, second trimester girl)

and accessory centers for the second and third metatarsal in 1 (control, first trimester boy).

3. *Rib Abnormalities.* Three types of rib abnormalities were found: cervical, lumbar, and short or rudimentary 12th dorsal. The incidence of these anomalies is shown in Table IV.

The over-all incidence was 20/163 (12.2 per cent), considerably higher than the 1:645 reported by Steiner[16] in his study of rib abnormalities in 38,105 roentgenograms of the spine, chest and abdomen on which ribs could be examined. Cushway and Maier[4] reported an incidence of 1:11 (9 per cent) supernumerary ribs in a study of 931 cases. Giles[5] found cervical ribs in 6.5 per cent, lumbar ribs in 6–8 per cent and rudimentary dorsal ribs in 8.73 per cent of 1,122 cases.

In this present study there was no significant difference in incidence of rib abnormalities between the "exposed" and the "control" children. The incidence was significantly higher among the girls, however. Of the total number of rib anomalies 4 consisted of lumbar ribs, 14 of rudimentary or short 12th dorsal rib, and 3 (all in females) of cervical ribs. No bipartite ribs, imbrication of ribs, or synostotic ribs were found.

TABLE IV

INCIDENCE OF RIB ABNORMALITIES

		Number Examined	Proportion with Rib Abnormalities			
			G.A. I	G.A. II	G.A. III	Total
Exposed	Boys	34	0/13	1/10	0/11	1/34
	Girls	38	2/8	2/14	3/16	7/38
	Total	72	2/21	3/24	3/27	8/72
Control	Boys	39	0/10	2/14	2/15	4/39
	Girls	52	3/17	5/18	0/17	8/52
	Total	91	3/27	7/32	2/32	12/91
Grand total		163	5/48	10/56	5/59	20/163

TABLE V

INCIDENCE OF VARIATIONS OF LUMBOSACRAL VERTEBRAE

		Number Examined	Proportion with Lumbosacral Variations			
			G.A. I	G.A. II	G.A. III	Total
Exposed	Boys	34	2/13	0/10	3/11	5/34
	Girls	38	1/8	1/14	2/16	4/38
	Total	72	3/21	1/24	5/27	9/72
Control	Boys	39	0/10	3/14	1/15	4/39
	Girls	52	1/17	4/18	3/17	8/52
	Total	91	1/27	7/32	4/32	12/91
Grand total		163	4/48	8/56	9/59	21/163

4. *Variations of Lumbosacral Vertebrae.* Aside from spina bifida occulta, sacralization of the fifth lumbar and lumbarization of the first sacral vertebra were the two variations seen in the roentgenograms of the lumbosacral region (Table v). Uncompensated variations in the number of the lumbar and sacral vertebrae were not found. No significant differences were demonstrated between boys and girls and between the exposed and control groups in the incidence of lumbosacral abnormalities.

Cushway and Maier[4] found sacralization of the transverse process of the fifth lumbar vertebrae in 50 out of 931 cases, an incidence of 1:18.6. Giles[5] quoted Sutherland's figure on 4.5 per cent in 12,000 cases and reported 9.2 per cent in his own series of 1,122. Brailsford found this variation either on one or both sides in 8.1 per cent of 3,000 persons. The incidence in this study was 21 in 163, or 12.9 per cent.

5. *Other Abnormalities.* Small single areas of cortical defect involved the distal portion of the femur in 4 cases and the proximal portion of the tibia in 1 case. In 2 boys, both exposed, the proximal epiphyses of the femur showed changes that roentgenologically suggested Legg-Perthes' disease.

6. *Multiple Abnormalities.* The data were

TABLE VI

OCCURRENCE OF MULTIPLE SKELETAL ABNORMALITIES

		Gestational Age on Aug. 9, 1945	No Anomalies	Single Anomalies	Multiple Anomalies
Boys	Exposed	I	6	3	4
		II	3	5	2
		III	2	5	4
	Control	I	2	6	2
		II	1	5	8
		III	3	6	6
Girls	Exposed	I	1	4	3
		II	2	9	3
		III	4	7	5
	Control	I	5	8	4
		II	4	8	6
		III	1	8	8

examined to determine (a) if the skeletal abnormalities described in the preceding paragraphs tended to occur in combinations rather than singly, and (b) if multiple anomalies were more frequent among the exposed children. From Table VI it was concluded that there was no tendency for the abnormalities to occur more often in combination than singly and more often in exposed than in control children.

7. *Comment.* In this investigation, the occurrence of the types of skeletal anomalies reported in experimentally irradiated mice and rats could not be demonstrated in children exposed at various stages of intrauterine existence to the atomic bomb. There was no difference between exposed and control children in the incidence of the more common bone variations. This survey, however, was conducted in 1951 and 1952 on children who were living six and seven years after exposure. It is quite possible that the more extreme degrees of abnormalities were present among the fetuses that were aborted or delivered dead and among infants that died in the early postnatal period. Data collected by Yamazaki, Wright and Wright[21] show that 4 abortions, 6 stillbirths, 6 neonatal deaths and 3 infant deaths occurred anong the exposed group. In contrast, 2 abortions, 1 stillbirth, 1 neonatal death and 3 infant deaths were reported among the control group.

In this study, an effort was also made to determine whether or not the presence of radiation symptoms in the mother was associated with increased incidence of skeletal abnormalities in the child. Although the numbers concerned were quite small, no correlation could be demonstrated.

SUMMARY

1. A roentgenographic survey of the skeletal system was made on 74 children who were exposed *in utero* to the atomic bomb explosion in Nagasaki, Japan at distances under 2,000 meters from the hypocenter. The findings were compared with those on a group of 91 children also exposed while *in utero* to the bomb but at distances of 4,000 to 5,000 meters.

2. No differences in the incidence of skeletal abnormalities were found between the two groups.

Carl A. Harris, M.D.
Committee on Atomic Casualties
National Research Council
2101 Constitution Ave.
Washington 25, D.C.

REFERENCES

1. BRAILSFORD, J. F. The Radiology of Bones and Joints. Williams and Wilkins Co., Baltimore, Md., 1948.
2. BUCY, P. C. In: McQuarrie and Brenneman's Practice of Pediatrics. W. F. Prior Co., Hagerstown, Md., Vol. IV, Chap. 15.
3. CAFFEY, JOHN. Pediatric X-ray Diagnosis. Year Book Publishers, Inc., Chicago, 1950.
4. CUSHWAY, B. C., and MAIER, R. J. Routine examination of spine for industrial employees. *J.A.M.A.*, 1929, *92*, 701–704.
5. GILES, R. G. Vertebral anomalies. *Radiology*, 1931, *17*, 1262–1266.
6. GIRDANY, B. R., and GOLDEN, R. Centers of ossification of skeleton. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1952, *68*, 922–924.

7. Hefke, H. W. Roentgenologic study of anomalies of hands in 100 cases of mongolism. *Am. J. Dis. Child.*, 1940, *60*, 1319–1323.
8. Hicks, S. P. Developmental malformations produced by radiation; timetable of their development. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *69*, 272–293.
9. Job, T. T., Leibold, G. J., Jr., and Fitzmaurice, H. A. Biological effects of roentgen rays; determination of critical periods in mammalian development with x-rays. *Am. J. Anat.*, 1935, *56*, 97–117.
10. Keith, A. Human Embryology and Morphology. Williams and Wilkins Co., Baltimore, Md., 1948.
11. Lewis, W. H. Gray's Anatomy. Lea & Febiger, Philadelphia, 1942.
12. Pryde, A. W. Personal communication.
13. Russell, L. B. X-ray induced developmental abnormalities in mouse and their use in analysis of embryological patterns. I. External and gross visceral changes. *J. Exper. Zool.*, 1950, *114*, 545.
14. Russell, L. B., and Russell, W. L. Radiation hazards to embryo and fetus. *Radiology*, 1952, *58*, 369–376.
15. Scammon, R. E. In: Morris' Human Anatomy. By Morris, H. Edited by Schaeffer, J. P. Tenth edition. The Blakiston Company, Philadelphia, 1942.
16. Steiner, H. A. Roentgenologic manifestations and clinical symptoms of rib abnormalities. *Radiology*, 1943, *40*, 175–178.
17. Sutow, W. W., and Ohwada, K. Standards for assessment of skeletal development among Japanese children. To be published.
18. Warkany, J., and Schraffenberger, Elizabeth. Congenital malformations induced in rats by roentgen rays; skeletal changes in offspring following single irradiation of mother. Am. J. Roentgenol. & Rad. Therapy, 1947, *57*, 455–463.
19. Warner, E. N. Survey of mongolism with review of 100 cases. *Canad. M.A.J.*, 1935, *33*, 495–500.
20. Wilson, J. G., and Karr, J. W. Effects of irradiation on embryonic development. I. X-rays on tenth day of gestation in rat. *Am. J. Anat.*, 1951, *88*, 1–33.
21. Yamazaki, J. N., Wright, S. W., and Wright, P. M. Study of outcome of pregnancy in women exposed to A-bomb in Nagasaki. Atomic Bomb Casualty Commission, December 18, 1952.

RADIUM THERAPY IN CANCER OF THE SKIN AND LIP*

By B. L. FEUERSTEIN, M.D.
BAY SHORE, NEW YORK

IT IS not the intent of this paper to prove that radium therapy is the treatment of choice, preferable to roentgen rays, in cancer of the skin. Until 1925, radium in the form of needles, gold seeds, plaques and molds was the commoner method used for skin cancer. However, in recent years, roentgen radiation has gradually replaced radium in most institutions; the vast majority of scientific papers and textbooks on the subject now stress roentgen irradiation as the preferred method. This paper intends to review a method of treatment which of late has undeservedly lost popularity with most radiologists. Since 1936, of 23 original articles on the treatment of cancer of the skin and lip in the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY and in *Radiology*, fourteen authors presented roentgen radiation as the principal treatment used; five used both roentgen radiation and radium; only four used radium exclusively. All of the last group were published over ten years ago.

Most young radiologists today complete their training with inadequate experience in the use of radium, having been taught that the knowledge of one modality is sufficient. To an extent, this is true; almost all cases of skin cancer can be treated by roentgen rays or radium. However, there are types of cases, examples of which will be shown, in which one of the two methods is preferable and can be expected to produce a superior end result. Such cases may be therapeutic problems if only roentgen radiation, or radium is available; if both modalities are available, the problem case may become a simple one.

The reasons usually advanced for preferring roentgen irradiation are (1) simplicity; (2) the avoidance of exposure of personnel to radium; (3) economy.

That roentgen irradiation is a simpler procedure than radium mold therapy is true for lesions near the eye. However, for other parts of the face, an accurately fitting wax mold can be made in about thirty minutes. This compares favorably with the time required to prepare a patient for a series of three to ten roentgen treatments as is usually prescribed. If contact radium is used, the time required is less than for roentgen therapy.

Roentgen irradiation insures freedom from exposure of personnel. However, tests have indicated that the amount of exposure while using radium for skin lesions is very small. In topical application, approximately 10 milliroentgens of body exposure is obtained from the time the radium is removed from the safe until it is replaced, of which half reaches the technician and half the physician. In making a mold, approximately 20 milliroentgens of exposure is involved. Assuming 300 milliroentgens to be a safe weekly exposure, few therapists will exceed this figure.

The economic reason advanced is that the same equipment used for deep roentgen therapy is adequate for the treatment of superficial cancer. Conventional deep roentgen therapy apparatus of 250 kv. (peak) will operate as low as 100 kv. with a half-value layer of about 3 mm. Al. On the other hand, the same radium used for cervical lesions is ideal for skin cancer. Furthermore, there is no depreciation of equipment to consider. Over a period of years, radium is definitely less expensive, requiring no calibration, upkeep cost, or replacement.

Interstitial radium therapy is a much more complicated procedure than either

* Presented before the Thirty-fourth Annual Meeting, American Radium Society, Chicago, Ill., June 8–10, 1952.

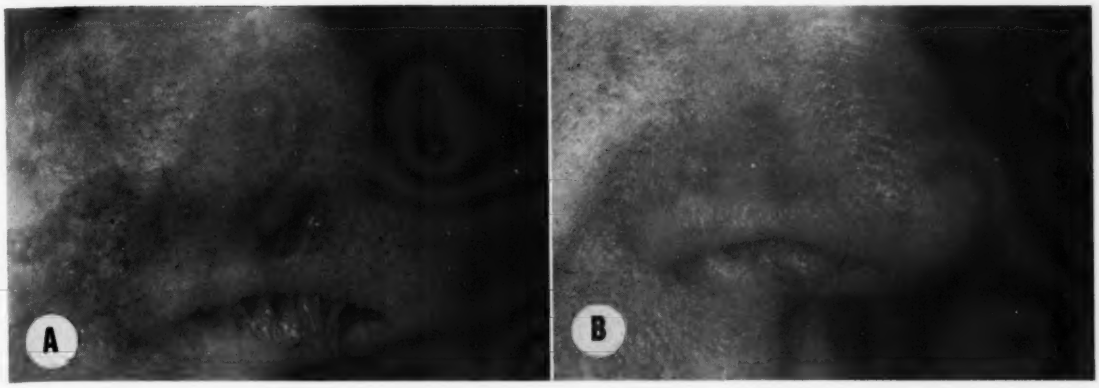

FIG. 1. (*A*) Small epithelioma of the skin of the nose. Suitable for contact radium therapy. (*B*) Lesion after treatment.

roentgen irradiation or surface radium therapy and of necessity exposes personnel to much more radiation. This will be discussed later.

In the treatment of skin cancer during the past twenty years, we have used both methods in almost every variety of lesion and have found advantages in each method. First, in our experience, radium leaves a better scar than roentgen therapy. Other writers have agreed with this finding. While the cancerocidal effect of radiation is not determined by the radiation quality, there appears to be a difference in the effect of radiation quality on normal tissue and its reparative ability. Second, we prefer radium where it is desirable to avoid a high depth dose. This is particularly true in lesions overlying the cartilage of the ear, despite recent articles indicating that roentgen therapy can be used in these areas with the same technique as is used in other parts of the skin. Radium is used for lesions on the bridge of the nose and of the lips overlying the junction of the lip and gum. Bleeding from the nasal septum is frequent after roentgen therapy, rare after radium. Protection of the gum at the alveologingival junction from roentgen radiation is often not possible; with radium, it is not necessary. Third, radium is the only method available when the patient cannot or will not leave his home; this is not uncommon in rural areas in aged individuals. Two such cases occurred in our practice recently. One was an eighty-five year old man with a small lip cancer; another, a ninety-five year old woman with a lip lesion 1 cm. in diameter,

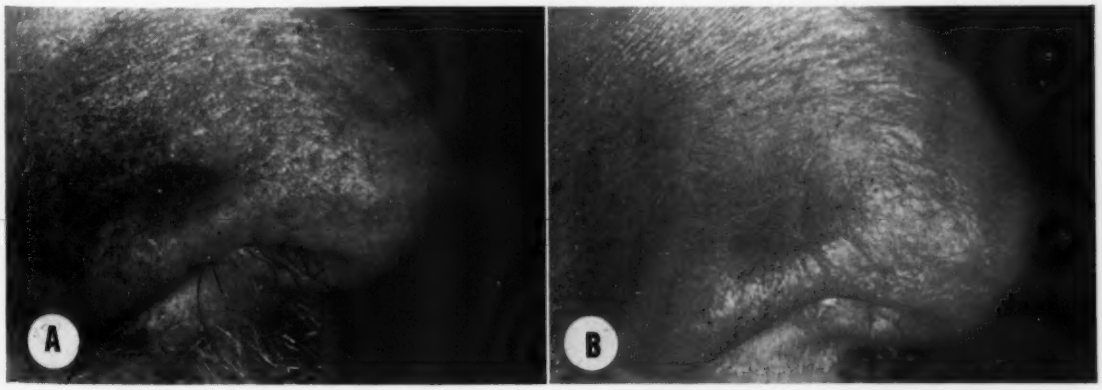

FIG. 2. (*A*) Epithelioma of the skin of the nose. Suitable for radium mold therapy. (*B*) Lesion after treatment.

FIG. 3. (*A*) Multiple epitheliomata of the skin of the nose. Lesion in the nostril accessible only by roentgen therapy. Lesion on the tip of the nose suitable for contact radium therapy. (*B*) Lesions after treatment.

growing rapidly. The man was treated with contact radium, the woman by gold seed implants. Both of these patients were practically bed-ridden and could not have been transported with safety for hospital or office treatment. A difficult therapeutic problem with roentgen therapy was simplified by the use of radium.

Roentgen therapy is preferred, first, in a large bulky lesion in which a high depth dose is necessary. In such cases, adequate dose in the depth by radium can be obtained only by implantation. Mold application in such cases is not usually practical. The necessary radium-skin distance is so great that too large an amount of radium is needed for ordinary use. Second, roentgen therapy is used in areas inaccessible to radium, such as lesions within the nostril. Third, roentgen therapy is used for lesions over 1 cm. in diameter near the eye. Using an intraocular shield, 98 per cent of the rays fail to reach the lens. Measurements with radium molds indicate that approximately 30 per cent reaches the lens. In twenty years at Bellevue Hospital, we have not had a single cataract following the use of radium; yet it does seem that when the dose reaching the lens can be reduced from 30 per cent to almost zero, the latter is to be preferred.

Radium treatment for cancer of the skin can be classified in three groups: (1) interstitial; (2) contact; (3) topical at a distance.

Interstitial radium therapy is rarely indicated in skin cancer. The operative procedure of puncturing the skin with needles or seeds should be avoided if possible and

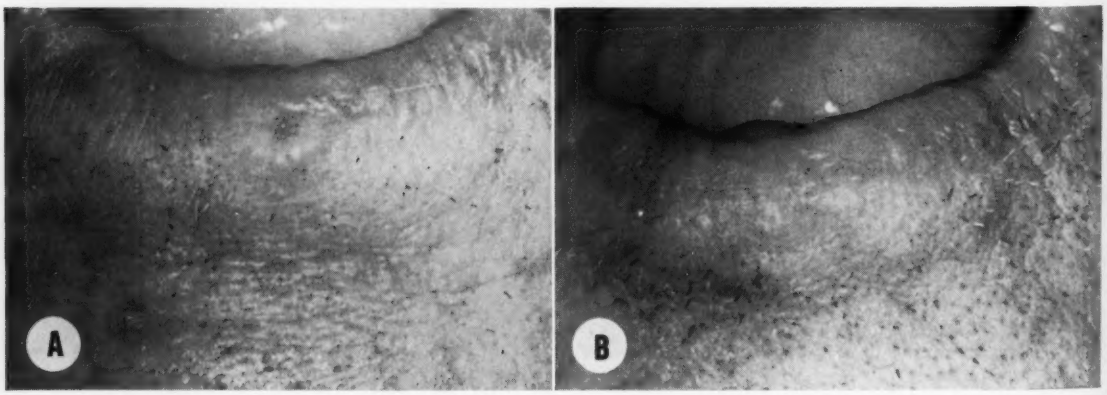

FIG. 4. (*A*) Small epithelioma of the lip. Suitable for contact radium therapy. (*B*) After treatment.

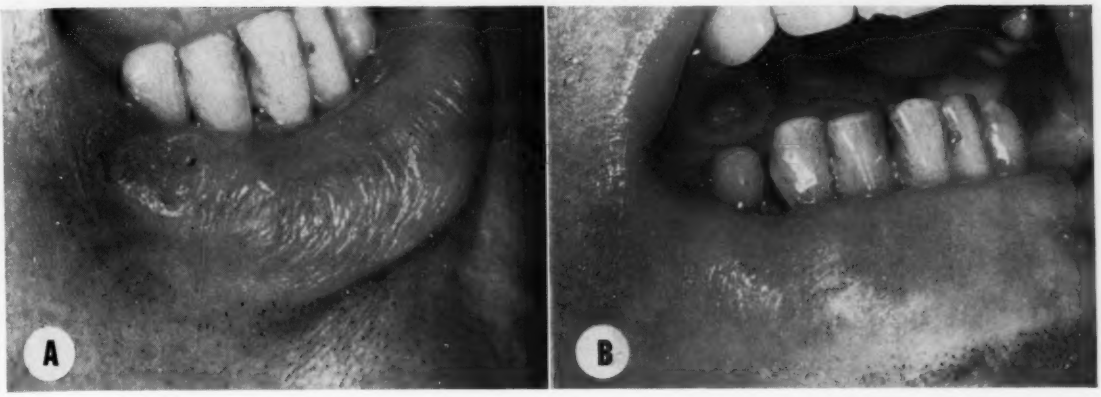

Fig. 5. (*A*) Epithelioma of the lip, too large for contact therapy, suitable for radium mold therapy. (*B*) Lesion after treatment.

reserved for those cases in which surface radium therapy is inadequate and if roentgen therapy is not available. Thus, elderly or uncooperative patients who cannot wear a mold for the requisite time of two or three days can be treated with gold seed implants at home. For large bulky or deeply infiltrating lesions, low intensity needles are implanted. Such lesions were common in the past; today, patients rarely delay treatment so long. Not over 1 per cent of skin cancers in our experience are today allowed to progress to this stage.

Lesions near the eye are also suitable for gold seed implants as the method gives an intensive dose within a very small area; the fall off in dosage is rapid enough to protect the cornea. With these few exceptions, we no longer use interstitial radium therapy.

Contact radium therapy, using platinum filtered tubes directly on the skin is a simple, adequate method for treatment of superficial skin cancer—lesions under 1 cm. in diameter, with no induration. If larger, thickened lesions are treated in this manner, surface healing will occur but recurrence will follow in a few months. During the past ten years, as a result of the intensive campaign to educate the public in the symptoms of cancer, skin lesions are seen by the doctor at a much earlier stage. Rarely do we see the extensive lesions which were once commonplace. At least 50 per cent of all lesions can be treated by contact radium.

Platinum filtered tubes (1 mm.) containing 10 or 15 milligrams of radium each are strapped directly on the skin with adhe-

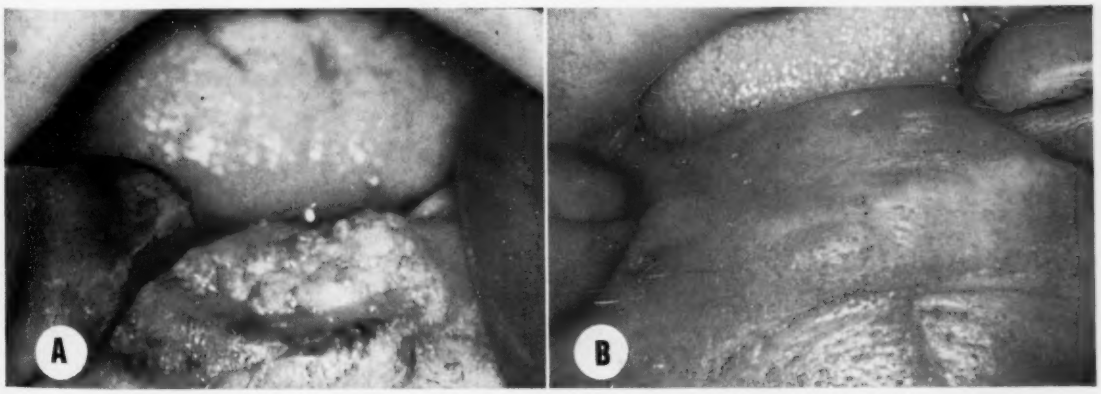

Fig. 6. (*A*) Extensive epithelioma of the lip involving more than half of the lower lip. (*B*) After treatment by radium mold therapy.

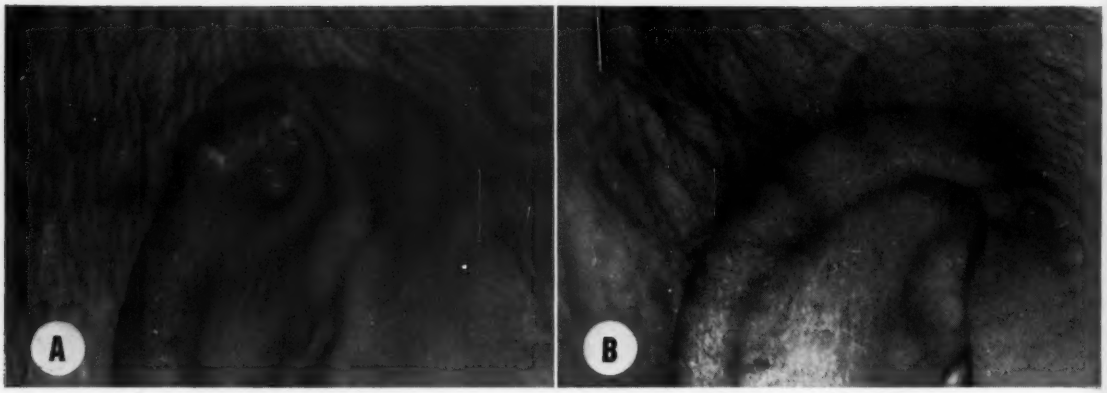

FIG. 7. (*A*) Epithelioma of the skin of the ear, ideal for radium mold therapy. (*B*) Lesion after treatment.

sive or Scotch tape. Two or three tubes, laid parallel and touching, are left in place for the requisite time for a dosage of 80 to 200 mg. hr. Because of the short distance, the dose is not calculated in gamma roentgens.

Larger lesions are treated with radium molds, using either dental or radon wax. The radon wax, which comes in blocks of 1 or 1.5 cm. thickness, is softened by immersion in hot water, not over 52° C. This softens the wax sufficiently to allow it to be molded over the involved area. Excessive heating will cause disintegration of the wax. By not exceeding the 52° C. temperature, the wax, although softened, will maintain its thickness. The radium tubes are then set into the surface of the wax away from the lesion and the mold tied in place. Radon wax is ideal for areas which are fairly flat, such as the cheek, temple, and forehead. In irregularly shaped areas, such as the lip, dental wax is preferred as it can be molded with greater ease. It is softened in hot water into a mass which is then molded over the lip, similar to a wax denture. The tubes are inserted as with radon wax. It must be remembered, however, that since dental wax is molded from a shapeless mass, its thickness is not fixed, hence the distance must be accurately measured after the mold is finished.

In making the molds, the radium tubes are placed in a square or circle according to the method of Paterson-Parker and the dosage calculated from their charts. This is between 5,000 and 6,000 r gamma. Experience and patience are essential in making the mold, so that the radiation is accurately directed. The excellent end re-

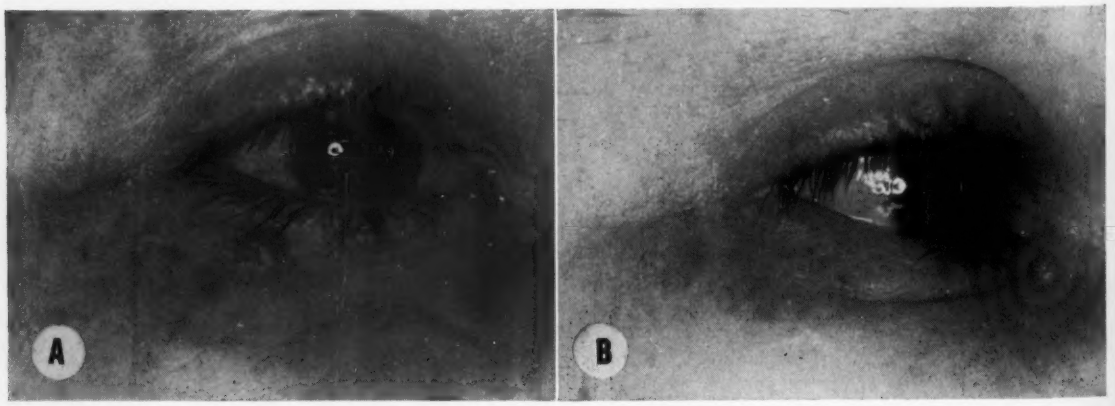

FIG. 8. (*A*) Small epithelioma of the skin near the eye, suitable for contact radium. (*B*) After treatment.

TABLE I

Location	No.	Modality	Under 2 yr.	Follow-up			
				2–3 yr.	3–4 yr.	4–5 yr.	Over 5 yr.
Nose	34	Contact 26 Mold 8	14 (1*)	4	6	3	7
Scalp	3	Contact 2 Mold 1	3				
Eye	15	Contact 12 Mold 3	2	4	1	2	6
Ear	12	Contact 4 Mold 8	4 (1†)			3	4
Cheek	25	Contact 14 Mold 11	6 (1†)	1	5	4	8
Forehead	20	Contact 12 Mold 8	6	4 (2*)	2	4	4
Lip	16	Contact 6 Mold 10	2 (1* 1‡)	5	1	2	2
Trunk	3	Contact 1 Mold 2		1 (1‡)			2
Chin	3	Contact 1 Mold 2	1		2		
Neck	7	Contact 2 Mold 5	1 (1†)		1	1	3
Hand	2	Contact 0 Mold 2	2 (1*)				
TOTAL	140	Contact 80 Mold 60	41	19	18	19	39

Pathology:	
Basal cell	78
Squamous	46
Basosquamous	7
Epithelioma	9
	140

* Died; no evidence of disease	5
† Lost; no follow-up	4
‡ Recurrence excised, well after 5 yr.	1

sults obtained with this method, especially the soft smooth scar with a minimum of late atrophy and telangiectasia, justify the extra effort expended.

Table 1 summarizes the results obtained in 140 consecutive cases treated with radium and proved by biopsy. It will be noted that 139 cases have remained free of disease. The single recurrence was in a patient with an epithelioma of the skin of the back developing in a sebaceous cyst. Surgery was refused and the patient was therefore treated with radium. There was a recurrence, at which time the patient agreed to surgery.

This very high percentage of cures can be explained. As previously stated, patients are appearing with lesions at a much earlier

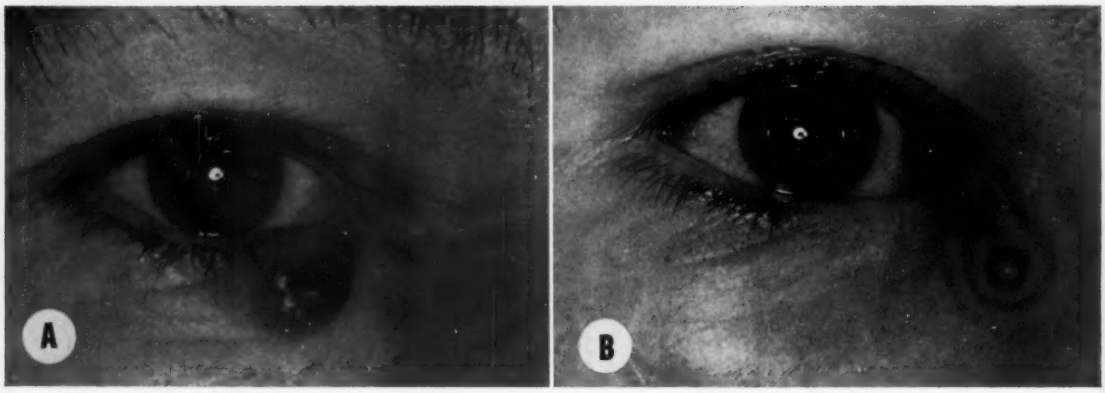

FIG. 9. (*A*) Epithelioma of the skin near the eye, suitable for roentgen therapy. (*B*) After treatment.

stage, due to the public awareness of cancer. Also, the patients in this group come from a better class of people, both economically and intellectually, and are prone to seek medical care early. Finally, as noted previously, very large indurated lesions were not treated by radium. This last factor is the least important; not more than one case in 100 that we see is referred for roentgen therapy solely for this reason.

SUMMARY

1. Most cases of cancer of the skin can be cured by roentgen rays or radium. However, treatment can often be simplified and better cosmetic results obtained in certain types of lesions if one is adept at both methods.

2. Radium therapy usually gives a better cosmetic result and is preferred where high depth dose is to be avoided and in small early lesions so frequently seen today.

3. Roentgen therapy is preferred in less accessible areas, such as near the eye and in large bulky lesions.

4. The end results of the treatment by radium in 140 cases are presented.

375 East Main St.
Bay Shore, New York

REFERENCES

1. BOGART, F. B. Irradiation treatment of cancer of skin. *Radiology*, 1941, *36*, 12–22.
2. DEL REGATO, J. A. Roentgen therapy of carcinoma of lower lip. *Radiology*, 1948, *51*, 499–508.
3. DRIVER, J. R., and COLE, H. N. Treatment of epithelioma of skin of ear. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *48*, 66–73.
4. EBERHARD, T. P. Treatment of epitheliomas of skin. *Radiology*, 1947, *49*, 620–626.

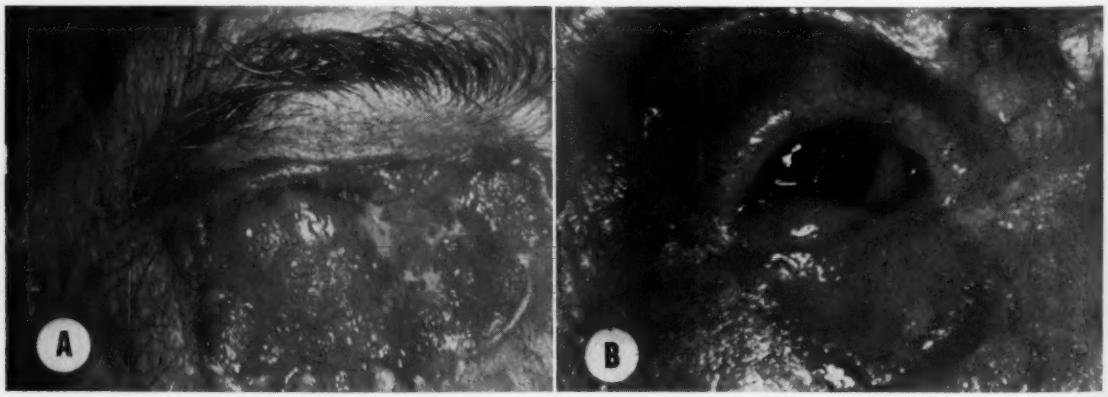

FIG. 10. (*A*) Extensive epithelioma of the skin overgrowing the eye, suitable for roentgen therapy. (*B*) Lesion three weeks after treatment.

5. Hale, C. H., and Holmes, G. W. Carcinoma of skin. *Radiology*, 1947, *48*, 563–569.
6. Howes, W. E., and LaRosa, F. J. Carcinoma of lower lip. Am. J. Roentgenol. & Rad. Therapy, 1942, *47*, 39–49.
7. Howes, W. E., and Rosenstein, J. Cancer of lower lip. Am. J. Roentgenol. & Rad. Therapy, 1948, *60*, 763–775.
8. Hunt, H. B. Treatment of large protruding carcinomas of skin and lip by irradiation and surgery. Am. J. Roentgenol. & Rad. Therapy, 1940, *44*, 254–264.
9. Kaplan, I. I. Radiation therapy of malignant lesions of lip. *Radiology*, 1937, *28*, 533–543.
10. MacComb, W. S. Low voltage roentgen therapy in skin cancer. Am. J. Roentgenol. & Rad. Therapy, 1939, *41*, 437–440.
11. Murphy, J. T., and Hufford, C. E. Use of 200,000 volts in treatment of advanced superficial cancer. *Radiology*, 1941, *36*, 23–33.
12. Pendergrass, E. P., Hodes, P. J., and Garrahan, C. J. Roentgen therapy by method of Chaoul. *Radiology*, 1939, *32*, 142–154.
13. Pendergrass, E. P., and Hodes, P. J. Further experiences with Chaoul therapy. *Radiology*, 1941, *37*, 550–559.
14. Perkins, C. W. Roentgen-ray treatment of skin cancer. *Radiology*, 1939, *33*, 191–195.
15. Pfahler, G. E. Treatment of cancer of lip and mouth. *Radiology*, 1940, *35*, 598–605.
16. Robinson, G. A., and Harris, J. H. Treatment of cancer of skin of nose. Am. J. Roentgenol. & Rad. Therapy, 1942, *48*, 59–65.
17. Schreiner, B. F., and Christy, C. J. Results of irradiation treatment of cancer of lip. *Radiology*, 1942, *39*, 293–297.
18. Sims, G. P. High-voltage roentgen irradiation of accessible cancer of skin and mucous membrane. *Radiology*, 1941, *37*, 666–672.
19. Ullmann, H. J. Cancer of skin. *Radiology*, 1946, *46*, 279–281.
20. Ward, C. B., and Betts, A. Epithelioma of lip. *Radiology*, 1936, *26*, 349–355.
21. Widmann, B. P. Cancer of lip. Am. J. Roentgenol. & Rad. Therapy, 1950, *63*, 13–24.
22. Wigby, P. E., and Cohen, M. Radium therapy of carcinoma of skin. *Radiology*, 1940, *35*, 70–78.
23. Wilkins, G. C. Methods of applying radium element in form of needles and tubes. Am. J. Roentgenol. & Rad. Therapy, 1938, *39*, 816–821.

EXPERIMENTAL ORBITOGRAPHY*

By P. THOMAS MANCHESTER, JR., M.D., JOSE BONMATI, M.D., TED F. LEIGH, M.D., *and* F. PHINIZY CALHOUN, JR., M.D.

EMORY UNIVERSITY, GEORGIA

ORBITOGRAPHY is defined as the roentgenographic contrast visualization of orbital structures.

Tumors within the orbit frequently are difficult to identify with any degree of certainty. The clinical and roentgenographic criteria commonly used in the identification of expanding lesions within this structure are not entirely satisfactory in many cases; such criteria include exophthalmos, diplopia, orbital and ocular calcifications, and changes in the orbital walls (erosion, destruction, hyperostosis).

To determine the usefulness of orbitography as a diagnostic procedure in humans, animal experiments were done, using rabbits and injecting a water soluble contrast medium (diodrast) directly into the orbits. It was intended from these experiments to obtain information regarding the following questions:

(1) What is the optimum amount of contrast medium needed for satisfactory visualization of the orbital structures on roentgenograms?

(2) Which is the space of choice within the orbit for the injection?

(3) How rapidly is contrast medium absorbed from the orbit?

(4) What effect does the contrast medium have on the delicate eye and on other orbital structures?

REVIEW OF THE LITERATURE

The literature on orbitography is relatively scant, most of the papers having originated in Europe.

In 1935 Katz and Ledoux[7] injected diodrast experimentally into the orbits of cats and into 8 humans (immediately preceding enucleation); these were specifically done to determine the length of the eye.

Marchesini and Ghio[9] reported in 1935 their results after the experimental injection of radiopaque media into the orbit of rabbits to outline the muscle cone. They found the oily media to be unsatisfactory. Lithium iodide was apparently good, but it caused considerable pain and was therefore discarded. With thorotrast the image was good; however, the authors reserved judgment because of the long permanence of this contrast medium in the soft tissues and its radioactivity.

In 1938 Last[8] injected CO_2 into the orbit of 6 patients outlining tumors by this means. However, subsequent reports by Godtfredsen,[4] and later by Grino and Billet,[6] doubted the value of the method with their cases. They concluded that gas is a poor contrast medium in the neighborhood of the air cells of the paranasal sinuses, though it retained its value for the identification of intraocular foreign bodies, when injected between the sclera and Tenon's capsule.

In Germany, in 1938, Thiel[5,11] satisfactorily outlined an orbital tumor by injection of thorotrast. In 1941, Glowatsky[3] reported the case of a patient with a suspected orbital tumor which was injected with thorotrast, following Thiel's method. Roentgenograms of the orbit showed the thorotrast to be collected in the eyelids, but not in the retrobulbar area. A large tumor was present in the orbit, preventing the thorotrast from filling the retrobulbar space. In these last 2 cases, the thorotrast remained at the site of the injection for a long period.

ANATOMY OF THE RABBIT'S ORBIT

The rabbit's orbit houses two important surgical spaces which are separated from each other by fascial planes: (1) the peripheral surgical space, and (2) the central

* From the Departments of Ophthalmology and Radiology, Emory University School of Medicine, The Grady Clay Memorial Eye Clinic, Grady Memorial Hospital, Atlanta, Georgia; and the Department of Radiology, Emory University Hospital, Emory University, Georgia.

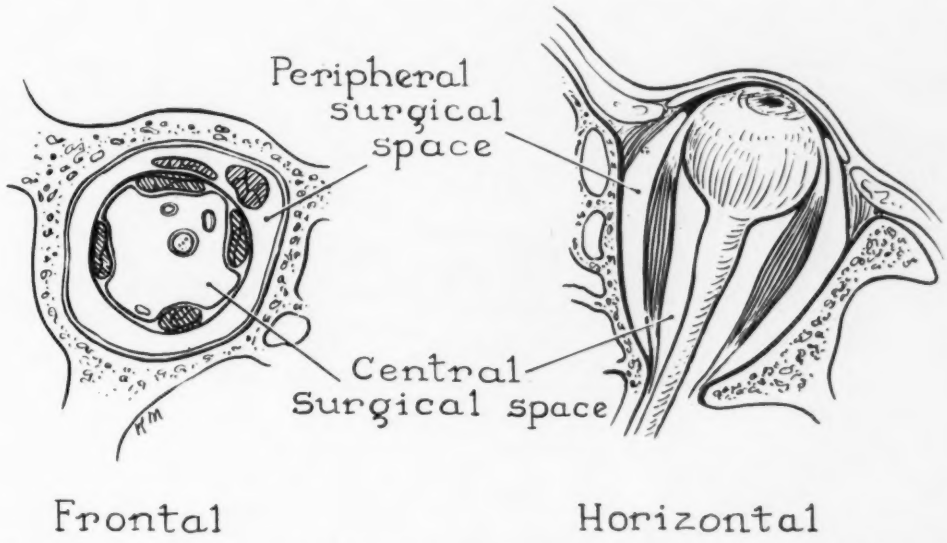

Fig. 1. Schematic drawing of sections through the rabbit's orbit, showing the peripheral and central surgical spaces.

surgical space, or muscle cone (Fig. 1). The muscle cone is formed by the four rectus muscles as they leave the eye and converge posteriorly to conjoin at the apex of the orbit. Between these muscles is a continuous sheath of fascia called the intermuscular septum, which is capable of retaining fluids. The muscle cone contains loose fat, a few blood vessels, and the optic nerve which is enclosed in its own protective dural sheath. The remainder of the orbit out to the periosteum is the peripheral surgical space.

The human orbit resembles that of the rabbit, but its volume is considerably larger (approximately five times). The human orbit diverges about 20 degrees from the sagittal plane, in contrast to the rabbit's orbit, which is close to 90 degrees; because of this, axial films of the human orbit correspond to lateral films of the rabbit orbit.

TECHNIQUE OF EXPERIMENTS

Two series of experiments were carried out. In both the rabbit was anesthesized with intravenous nembutal. Pontocaine was instilled into the conjuctiva. The superior rectus muscle was grasped with forceps to put Tenon's fascia on a stretch and a small curved needle was inserted between the superior oblique muscle and the eye. Initially roentgenograms were taken immediately after the injection of the contrast medium in frontobasal, lateral, and oblique positions. The oblique position proved to be the best by eliminating superimposed bony structures and so in our later experiments this was the only projection used. In some cases delayed films were made to study the absorption rate of the contrast medium. External examination of the eye and its adnexa, and funduscopic examination of the rabbits injected was carried out in the following days. Some of the rabbits were sacrificed at varying intervals after the injections and their orbital tissues studied microscopically.

In the first series of experiments only, 35 per cent diodrast was injected in one of the orbits of the rabbit. A total of 14 injections in 13 rabbits was performed.

In the second series hyaluronidase was also used. It is well known that this substance promotes diffusion of different media in soft tissues. Simon and Narins[10] have reported a more rapid absorption of subcutaneously injected contrast media following the use of hyaluronidase. To obtain the maximum effect they recommended the use of 15 or more turbidity units of hyaluronidase at least twenty minutes before the injection of the contrast media. Byrne and Melick,[1] experimenting with guinea pigs, also found such an action of hyaluronidase; they used it subcutaneously in 25 infants

FIG. 2. (*A*) Oblique and (*B*) lateral roentgenograms of the rabbit's orbit following the injection of 1.0 cc. of diodrast in the peripheral surgical space.

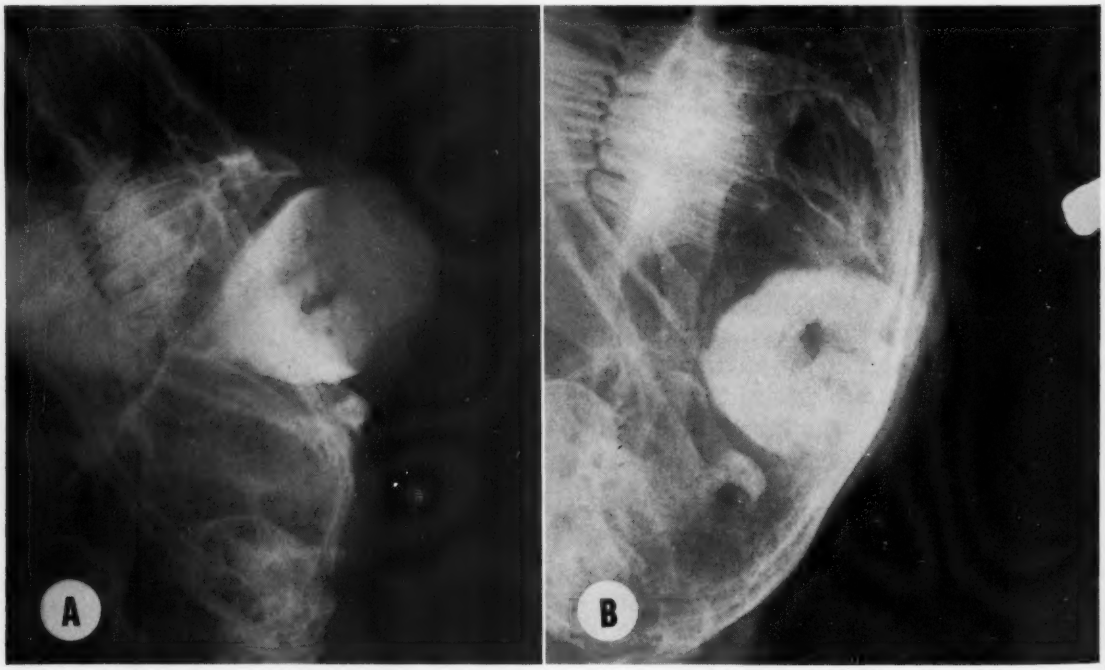

FIG. 3. (*A*) Oblique and (*B*) lateral roentgenograms of the rabbit's orbit following the injection of 1.0 cc. of diodrast within the muscle cone.

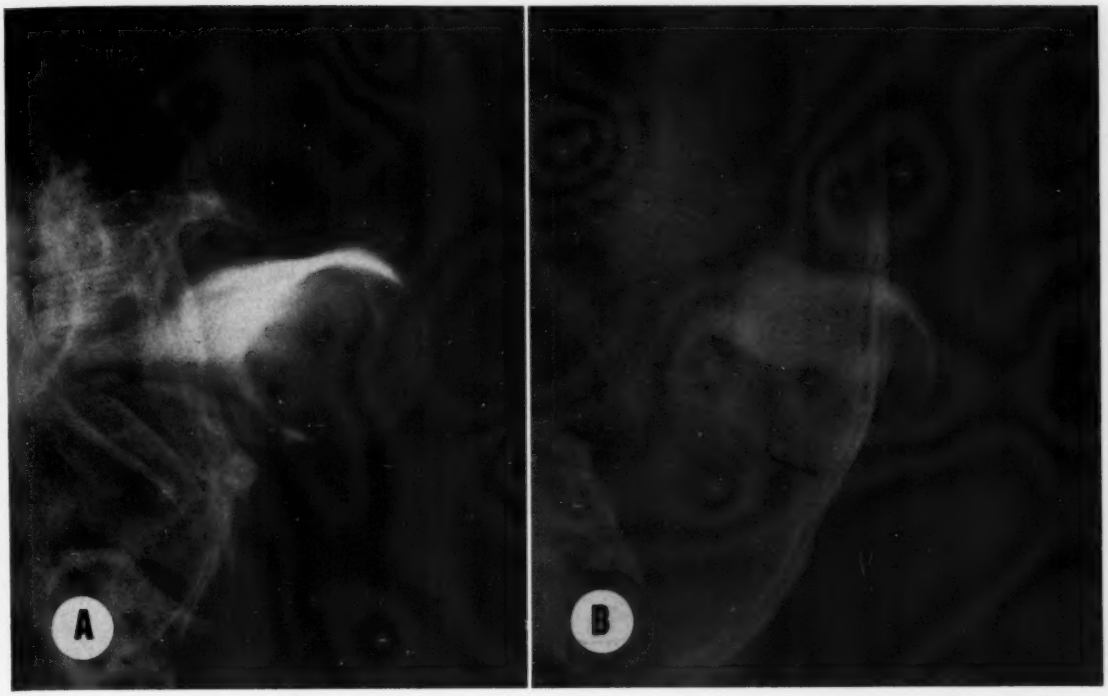

Fig. 4. (*A*) Oblique and (*B*) lateral roentgenograms of the rabbit's orbit following the injection of latex and 1.0 cc. of diodrast. Note the filling defect in the muscle cone.

immediately preceding the injection of 70 per cent urokon for excretory pyelography. In our studies, from 15 to 50 TRU* of a commercial preparation of hyaluronidase ("wydase") was used. Both orbits of 11 rabbits were injected. In 10 rabbits, one orbit received 1 cc. of 35 per cent diodrast and the opposite orbit was injected with hyaluronidase and 1 cc. of diodrast. In the remaining rabbit, both orbits received hyaluronidase, following which one orbit was injected with 1 cc. and the opposite with 2 cc. of 35 per cent diodrast. In 6 cases the injection of hyaluronidase preceded the one of diodrast by an interval of twenty or more minutes; in the remaining 5 cases no interval between the injections was allowed.

RESULTS OF EXPERIMENTS

1. *Optimum Space for Injection.* As mentioned earlier, there are two anatomical spaces within the orbit—the peripheral and the central (muscle cone) surgical spaces. When the peripheral space is injected, the contrast medium tends to collect in a shapeless mass, which lacks any diagnostic qualities (Fig. 2, *A* and *B*). When the central space (or muscle cone) is injected, the medium spreads through it, assuming a cone shape with base around the posterior periphery of the eye and apex near the optic foramen. In the lateral film it appears as a circular image and in the oblique film as a triangular one (Fig. 3, *A* and *B*). Theoretically a mass within the orbit will distort this opacified muscle cone, and thus produce films of diagnostic quality. This was verified in one instance when latex rubber was injected into the orbit prior to the injection of the contrast medium; it produced a mass which deformed the muscle cone (Fig. 4, *A* and *B*).

2. *Amount of Contrast Medium.* Injection of 0.5 cc. of diodrast into the central space of the orbit of the adult rabbit inadequately filled the muscle cone, and the films obtained were not of diagnostic quality. With 1.0 cc. the entire cone filled well, and in some instances, the negative shadow of the optic nerve was also apparent in the lateral film. Not uncommonly, a fine opaque line paralleling the outer border of the muscle cone was visible on the roentgenogram; this was caused by the outward bulge of the fascia connecting the muscle. When 1.5 cc., 2 cc., and 4 cc. were injected,

* TRU = Turbidity Reduction Unit.

FIG. 5. Oblique roentgenogram of the rabbit's orbit following the injection of 4.0 cc. of diodrast.

the muscle cone did not show any greater opacity than when 1.0 cc. was used, but did show greater distortion because of more marked bulging of the fascia. Also some of the contrast medium at times escaped from the muscle cone into the subconjuctival tissues, and this, too, produced confusing shadows on the roentgenogram (Fig. 5).

When hyaluronidase was used, the opacification of the muscle cone by the contrast medium was not as intense nor were its borders as sharply defined as when diodrast alone was used, especially when an interval between the injections was allowed. It was remarkable that, although great care was taken to avoid intravenous injections, the usual opaque shadow was very poor in 3 cases and no contrast medium was visible in 2 cases.

3. *Time of Disappearance of the Contrast Medium.* In 4 instances in the first series of experiments serial films of the orbit were made at thirty minute intervals following the injection of 1 cc. of diodrast. These showed only slight absorption of the medium within the first hour. Even after three hours, a faint opacification was still present (Fig. 6, *A*, *B*, *C* and *D*).

In the second series of experiments, when a short interval elapsed between the injection of hyaluronidase and diodrast *there was complete disappearance of the contrast medium from both orbits within the first thirty to ninety minutes in all the cases recorded*, even in the orbits in which the contrast medium alone was injected. Therefore, there was an absorption hastening effect attributable to this substance (Fig. 7, *A* and *B*). A satisfactory explanation for the action of hyaluronidase on the opposite orbit has not been found.

When the interval between the injections of hyaluronidase and diodrast was not observed, a faint opaque shadow was still present in the ninety minute film in all cases recorded. Although the absorption hastening effect was not so marked as when the interval was allowed, there was still some effect as compared with the time of disappearance of diodrast in the first series.

4. *Gross and Microscopic Changes in the Eye and Adnexa Following the Injections.* The gross and microscopic changes in the eyes and orbits following injection of the contrast media in the first series of experiments are summarized in Tables I and II. All rabbits receiving 1 cc. of diodrast showed immediate changes which apparently cleared within forty-eight hours; but on histologic studies of several of the exenterated orbits, necroses in the sclerae were observed (Fig. 8). As a control, 2 rabbits were injected with 1 cc. of saline each; neither of these showed any histologic changes on microscopic examination. All rabbits injected with 2 or more cc. of diodrast showed severe changes in the eyes and adnexa by inspection, and on microscopic examination, the orbital tissues revealed severe damage.

In the second series of experiments, the immediate changes in the eyes and orbits of the injected rabbits were strikingly less in degree than those in the first series (Table III). All five orbits examined microscopically were negative except in 1 case in which there was a slight hemorrhage and subacute inflammatory reaction twenty-four hours after the injection (Rabbit 21); in this case there had been no interval between the injection of hyaluronidase and diodrast, and the time of disappearance of diodrast had been greater than ninety minutes.

TABLE I

GROSS CHANGES FOLLOWING INJECTION OF DIODRAST

Rabbit	Orbit	Amount of Diodrast	Immediate Changes	Changes after 24 Hours	Changes after 48 Hours
1	Right	0.5 cc.	Miosis, slight edema of conjunctiva	None	None
1	Left	1 cc.	Miosis, slight edema of conjunctiva	Slight edema of conjunctiva and lid	None
6	Right	1 cc.	Miosis, slight edema of conjunctiva	Slight edema of conjunctiva and lid	None
7	Right	1 cc.	Miosis, questionable exophthalmos, slight edema of conjunctiva	Chemosis, moderate lid edema (sacrificed)	
11	Right	1 cc.	Essentially none	None	None
12	Left	1 cc.	Miosis	Cornea hazy, mild lid edema. Redness of conjunctiva	None (sacrificed)
13	Right	1 cc.	Miosis	Slight swelling and redness of conjunctiva	None
5	Right	1 cc.	Chemosis of conjunctiva	None	None
8	Left	1.5 cc.	Miosis, edema of conjunctiva, slight exophthalmos	Moderate lid edema, slight redness of conjunctiva, miosis, two retinal hemorrhages	Subsiding lid edema and conjunctival redness
2	Right	2 cc.	Miosis, exophthalmos, edema of conjunctiva, poor closure of lids	Not examined	Animal febrile, poor lid closure, grayness of cornea (sacrificed)
4	Right	2 cc.	Conjunctiva and lid edema, exophthalmos, poor lid closure	Exophthalmos, marked lid edema, multiple preretinal hemorrhages (sacrificed)	
25	Right	2 cc.	Miosis, exophthalmos, poor lid closure	Marked lid edema, no definite fundus changes but many small gray dots seen	Subsiding edema, slight redness of eye, fundus unchanged
26	Left	2 cc.	Miosis, exophthalmos, poor lid closure	Marked lid edema, no definite fundus changes, but many small gray dots seen	Subsiding edema, fundus unchanged
27	Left	4 cc.	Miosis, edema of adnexa, conjunctival chemosis, poor lid closure	Marked edema of adnexa, conjunctival chemosis, no definite fundus changes	Marked, but subsiding edema, fundus unchanged

TABLE II

MICROSCOPIC CHANGES FOLLOWING INJECTION OF DIODRAST

Rabbit	Orbit injected	Amount of Diodrast	Autopsy after	Microscopic Findings
1	Right	0.5 cc.	4 wk.	Negative
7	Right	1 cc.	24 hr.	One small perforation of the sclera with some intraocular hemorrhage. In isolated areas fat necrosis seen.
12	Left	1 cc.	48 hr.	One small area in the sclera is acellular, which may be the site of a partial necrosis.
6	Right	1 cc.	2 wk.	Small scar over a site of perforation of the sclera.
1	Left	1 cc.	4 wk.	Negative
8	Left	1.5 cc.	2 wk.	A 2 mm. scleral perforation posteriorly. The interior of the eye is not involved otherwise.
4	Right	2 cc.	24 hr.	Severe swelling and discoloration of tissues. Eyelids edematous and red. Fat necrosis is considerable in some areas. Marked infiltration of all tissues with inflammatory cells. Scleral perforations with involvement of intraocular structures. Microscopically, the only normal tissue in the slides is the optic nerve. In summary: Orbital cellulitis and necrosis of the sclera with perforation of the globe.
2	Right	2 cc.	48 hr.	The histologic picture is essentially the same as in rabbit #4 but the changes are more marked. In addition, there is thickening of the cornea and a ring abscess.

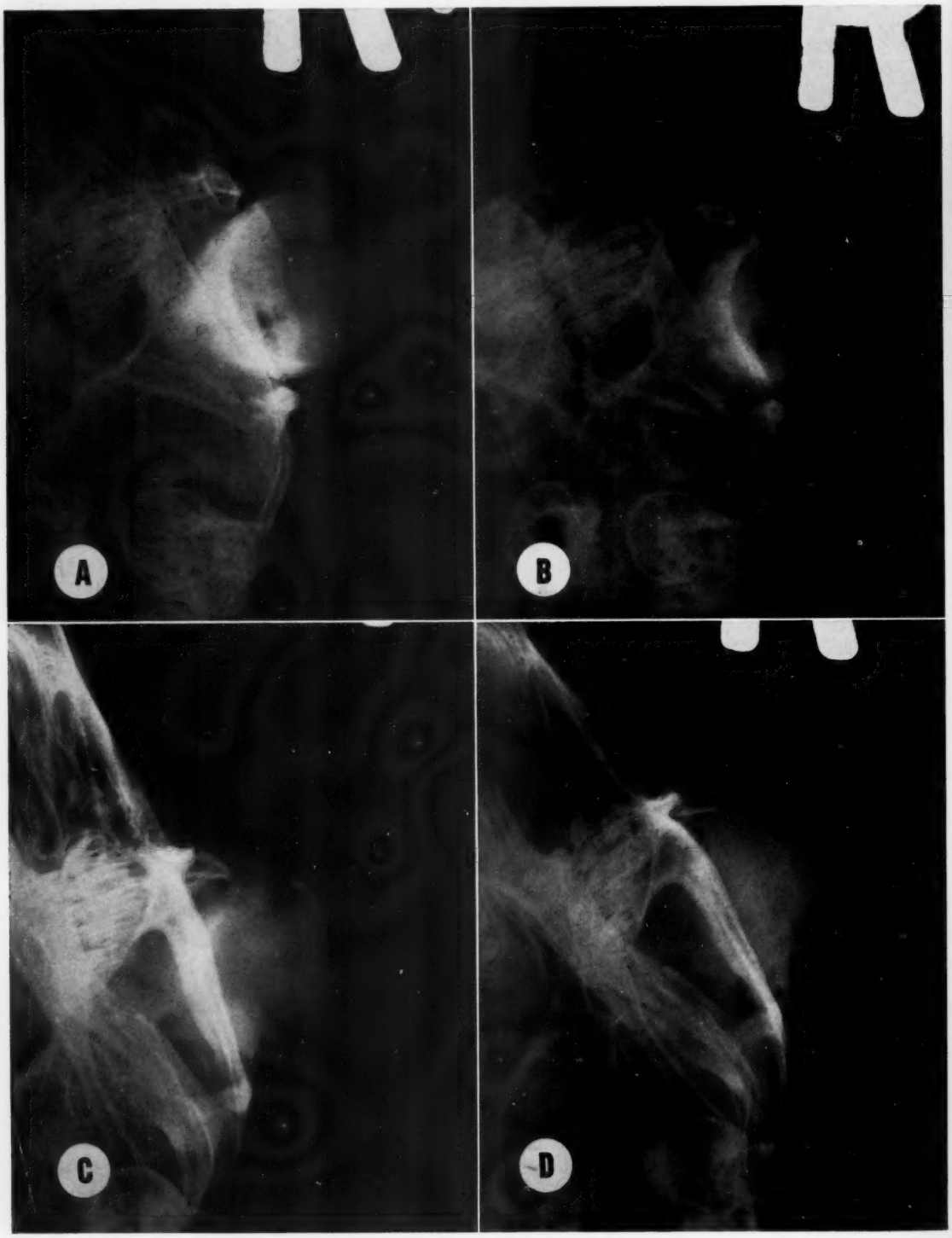

FIG. 6. (*A*) (*B*) (*C*) and (*D*). Roentgenograms of the rabbit's orbit 30, 60, 120 and 190 minutes after the injection of 1 cc. of diodrast.

Fig. 7 Roentgenograms of a rabbit's orbits (*A*) immediately and (*B*) 60 minutes after the injection of 1.0 cc. of diodrast. Hyaluronidase was injected 20 minutes prior to diodrast, in the right orbit. There is complete disappearance of the contrast medium from both orbits in the 60 minute film.

DISCUSSION

Several important conclusions can be drawn from the present experiments.

Fig. 8. Photomicrograph of the wall of the eye, 24 hours after the orbital injection of 1 cc. of diodrast. There is necrosis of the sclera (arrow).

The space of choice for injection of the contrast medium is the muscle cone. Its central location and its characteristic configuration are most useful in demonstrating any expanding lesion in the orbit. The looseness of the fat tissue in this space is advantageous for an easy spread of the contrast medium. The presence of the optic nerve in the muscle cone is not a disadvantage since it is well protected against damage by its hard dural sheath. It is likely that the muscle cone will offer similar advantages in human orbitography.

One cubic centimeter of diodrast produces in the rabbit films of satisfactory diagnostic quality. The use of larger amounts of contrast medium increases the risks of damage to the orbital structures and does not improve the quality of the roentgenograms. As the human orbit is about five times the volume of the rabbit's orbit, it may be assumed that 5 cc. of diodrast would give in humans satisfactory opacification of the muscle cone.

The time of disappearance of diodrast from the orbit is reduced considerably with the use of hyaluronidase. The absorption hastening effect of hyaluronidase is more marked when injected twenty minutes prior to the contrast medium; its effect is also apparent even if no interval between the injections is allowed.

TABLE III

GROSS AND MICROSCOPIC CHANGES FOLLOWING INJECTION OF DIODRAST AND HYALURONIDASE

Rabbit	Orbit	Media Injected	Immediate Changes	Changes 24 Hours after	Changes 48 Hours after	Autopsy after	Histologic Findings
14	Right	H.D.*	Slight mydriasis	None	None		
	Left	D	None	None	None		
15	Right	H.D.*	Slight mydriasis	None	None		
	Left	D	Slight miosis	Minimal edema of lid and conjunctiva	None		
16	Right	H.D.*	Slight mydriasis	None	None		
	Left	D	None	None	None		
17	Right	H.D.*	None	None	None	1 wk.	Negative
	Left	D	Slight miosis	None	None		
18	Right	H.D.*	Slight mydriasis	None	None		
	Left	D	Slight miosis	None	None		
19	Right	H.D.†	None	None	None	48 hr.	Negative
	Left	D	Slight edema of conjunctiva	None	None		
20	Right	H.D.†	None	None	None		
	Left	D	Slight edema of conjunctiva	None	None		
21	Right	D	Subconjunctival hemorrhages	Subconjunctival hemorrhages			
	Left	H.D.†	None			24 hr.	Some red blood cells in orbital tissues. Mild subacute inflammatory reaction. Normal eye and optic nerve
22	Right	H.D.†	None	None	None		
	Left	D	None	None	None		
23	Right	H.D.†	None	None	None		
	Left	D	None	None	None		
24	Right	H.D.*	None	None		24 hr.	Negative
	Left	H.D.‡	Some contrast media bulged beneath conjunctiva	None		24 hr.	Negative

* Interval of 20 minutes or more between injection of diodrast and hyaluronidase.
† No interval between injection of diodrast and hyaluronidase.
‡ Two cubic centimeters of diodrast were injected in this orbit.

The injection of diodrast alone causes damage to the orbital structures and the eye, and this damage is especially serious because of the importance of the organs involved.

For an understanding of the reactions

several factors are to be considered. First, the orbit is a closed space and its volume is definitely limited. If more than a certain amount of fluid is injected the circulation is impaired. There are no lymphatics in the in the orbit[2] and, surprisingly enough, it is poorly supplied with blood; this is in sharp contrast to the eye and the eyelids which are quite vascular. The orbit contains a large amount of fat; this fat is subject to necrosis, which actually was found in some of our cases.

The second problem centers around our injection medium. Thirty-five per cent diodrast is irritating and produces mild local reactions when injected in soft tissues. Tissue damage varies in proportion to the concentration of the contrast medium, and to the length of time that cells are exposed to this irritant. It has been found in our experiments that the degree of damage is considerably increased when more than 1 cc. of diodrast is used. Hyaluronidase, by increasing the absorption rate of diodrast from the orbit, reduces the time during which circulatory disturbances may take place. For such an action, hyaluronidase must be injected preceding the injection of the contrast medium. Under these circumstances the use of hyaluronidase reduces to a minumum the irritating effects of the latter and thus makes a safe method for investigation of orbital masses.

Orbital tumors are often suspected, but are inconclusively diagnosed because of their deep location and inaccessibility to direct means of exploration. There is needed a method for a more accurate diagnosis. This is illustrated by the fact that several authors have employed thorotrast injections into the orbit in an attempt to outline a mass. Their sometimes successful results regarding the presence and location of the tumor were overcome by the dangers of this contrast medium, which is retained indefinitely in the organism, and is weakly radioactive. The general use of thorotrast has been largely discarded in all fields of roentgenology.

A roentgenographic method for detection of orbital tumors by using a soluble contrast medium (diodrast) and by utilizing the proved absorption hastening properties of hyaluronidase has been developed experimentally. The safety of the procedure has been demonstrated in rabbits. It is intended to employ it cautiously in humans.

Ted F. Leigh, M.D.
Emory University Hospital
Emory University, Georgia

REFERENCES

1. Byrne, J. E., and Melick, W. F. Subcutaneous urography: description of new method utilizing 70 per cent urokon and hyaluronidase (preliminary report). *Urol. & Cutan. Rev.*, 1951, *55*, 193–199.
2. Elder, D. Textbook of Ophthalmology, C. V. Mosby Co., St. Louis. Vol. I, p. 188.
3. Glowatsky, F. Ueber Orbitographie in einem Falle von retrobulbarem Tumor. *Klin. Monatsbl. f. Augenh.*, 1941, *106*, 575–582.
4. Godtfredsen, E. Studies on orbital tumours; nature and incidence of orbital tumours. *Acta ophth.*, 1947, *25*, 279–293.
5. Grashey, R. Die bösartingen Geschwülste des Auges und seiner Umgebung. Hrsg. v. Rudolf Thiel. Bücherei des Augenarztes. Heft 6. Ferdinand Enke, Stuttgart, 1939.
6. Grino, A., and Billet, E. Diagnosis of orbital tumors by angiography. *Am. J. Ophth.*, 1949, *32*, 897–911.
7. Katz, D., and Ledoux, A. C. Measurement (roentgenometry) of antero-posterior diameter of eyeball in situ correlated with micrometer measurement following enucleation. *Am. J. Ophth.*, 1935, *18*, 914–924.
8. Last, M. Contrast media in orbital roentgenography. *Arch. Ophth.*, 1938, *19*, 1031.
9. Marchesini, E., and Ghio, A. Indagine radiografica della piramide orbitaria mediante l'introduzione di sostanze x opache. *Ann. di ottal. e clin. ocul.*, 1935, *63*, 662–675.
10. Simon, N., and Narins, L. Effect of hyaluronidase on absorption of subcutaneously deposited radiopaque substance. Am. J. Roentgenol. & Rad. Therapy, 1949, *61*, 91–94.
11. Thiel, R. Diagnosis of vascular anomalies and tumors in orbital fossa. German Ophthalmological Society. 52nd session, Heidelberg, July, 1938.

ROENTGEN-RAY AND ELECTRON RADIOGRAPHY OF THIN SPECIMENS*

By ARTHUR I. BERMAN *and* JAMES W. DUTLI
University of California, Los Alamos Scientific Laboratory
LOS ALAMOS, NEW MEXICO

I. INTRODUCTION

THE principles of radiography have been applied for many years to the study of thin structures in biology and metallurgy. The advantage over ordinary photographic methods is that components which are nearly identical in light absorption properties (even opaque) may be differentiated in the radiograph because of their dissimilar roentgen-ray and electron absorption properties. This fact often outweighs the main limitation of the radiographic technique, namely, low resolution. Where high resolution is required, roentgen-ray microscopy, as developed by Kirkpatrick[1] at Stanford, or electron microscopy —both differing from the methods discussed in this paper by providing primary magnification through a focusing action— may be employed.

II. ROENTGEN-RAY RADIOGRAPHY

A. Technique

In roentgen-ray radiography, roentgen rays from a tube with a small focal spot target, excited at low voltage (5 to 25 kv., peak), are incident on the specimen and fine-grain emulsion which are in intimate contact. The roentgen rays traverse the specimen in relative amounts depending upon its density, thickness, and especially its effective atomic number, producing a latent image on the emulsion, which then is developed routinely in the darkroom.

B. Resolution

The obtainable radiographic resolution depends upon three factors: (1) the emulsion grain size, (2) the amount of diffusion caused by scattered radiation, and (3) the focal spot size and target-specimen-emulsion geometry.

(1) In general, the limitation due to grain size is the most serious. The finest-grained emulsions manufactured at present are the Gaevert-Lippman and Eastman 649-GH and 548-O types.* The 649-GH plates are capable of resolving lines spaced 1 micron apart. This permits a magnification of 100 diameters before an image spacing of 0.1 mm. is reached, which is the widely accepted value of the minimum resolvable distance of the unaided eye when viewed at a normal distance of 25 cm. One can appreciate the inherent limitation of the radiographic technique with regard to resolution by comparing this value with the maximum permissible magnification, on this standard, of the light microscope of 2,000 diameters.

(2) The magnitude and influence of scattered radiation are unimportant in low-energy radiography. As the roentgen rays pass through matter they are diminished in intensity, or deviated in direction by electron interactions. The proportion of roentgen-ray quanta deviated, or scattered, to those absorbed is small; also, the scattered quanta have a high probability of being absorbed before leaving the material. Some fraction of the energy of a quantum which ejects a photoelectron may reappear, as the excited atom returns to its stable state, in the form of a quantum of low-energy fluorescent radiation, or other electrons called Auger electrons, which are readily absorbed. Thus the primary radiation activating the emulsion is accompanied

* A new metal-diazonium light-sensitive surface, developed at the Philips Laboratory in Holland, may equal or surpass these emulsions in resolving power.[2]

* This work was performed under contract with the Atomic Energy Commission.

by a small amount of roentgen-ray and secondary electron emission which contribute a relatively slight diffusion to the primary image. (The background resulting from scattering by intervening material, *e.g.*, air, or by rays emanating from parts of the target surrounding the focal spot, is negligible.)

(3) The limitation on resolution due to geometrical factors is usually less important than that due to the emulsion grain size. For thick specimens, or those in poor contact with the emulsion, the finite roentgen-ray source produces a penumbra which exceeds the minimum resolvable distance limited by emulsion grain size. From simple considerations one can see that if two points in the specimen are separated by d, and their images just overlap at distance s from the points to the emulsion, then $d=rs$, where r is the ratio of the longest dimension of the projected focal spot to the distance between the focal spot and emulsion. Thus, a 2 mm. diameter projected focal spot at 10 cm. from the emulsion will permit a lack of object-emulsion contact of 50 microns before an inherent minimum resolvable distance of 1 micron is reached. Both factors can contribute simultaneously to the diffusion of the image. The geometrical limitation may be minimized by: (a) reducing the distance between each point in the object and the emulsion, (b) decreasing the focal spot size, and (c) increasing the target-emulsion distance.

(a) The specimen should be sectioned as thinly as conventional preparation equipment, *e.g.*, microtome, will permit. Pressure should then be applied to provide uniform contact with the emulsion. This is best accomplished with a vacuum cassette[3] (a Los Alamos model is shown in Figure 1) in which an opaque Wratten infra-red filter (No. 87) is forced, by atmospheric pressure, into contact with the specimen and the sensitized surface. At extremely low operating voltages the cassette may have to be abandoned, and the exposure is then made in darkness in order to avoid filtration of the soft components and impractical exposure times. At still lower voltages the exposure must be made *in vacuo*.

Fig. 1. Vacuum cassette with plate and specimen in place.

(b) The focal-spot size can be decreased by suitable electron optical design of the tube electrodes, but this is usually accomplished at the expense of the total permissible roentgen-ray output. Focusing the electron beam at a minute target area places severe demands on the heat dissipation of the tube, necessitating a lowered tube current at the operating voltage desired. Special designs employing high-pressure fluid cooling, relative motion between target and focal spot, or a combination of both[4] can be used to alleviate this difficulty.

In some tube designs, the electron beam can, in principle, be focused to a spot as small as 100 A in diameter. Enlarged shadowgraphs can then be made directly, at the sacrifice of short exposure times, by placing the specimen close to the spot and far from the emulsion.[5]

(c) Increasing the target-emulsion distance presents practical difficulties of space limitation, and, especially, an increase in required exposure time resulting from the R^{-2} exp $(-\mu R)$ dependence of intensity (where R is the target-emulsion distance and μ is the air linear absorption coefficient).

Since the emulsion speed usually is an inverse function of its resolution, one ought to consider carefully the extent to which

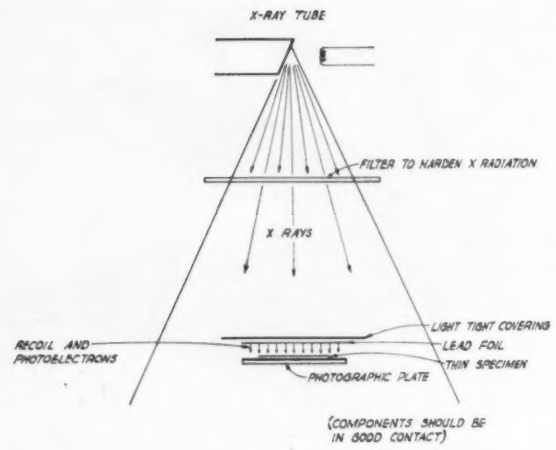

FIG. 2. Procedure for making electron radiographs.

proposals (a), (b), and (c) should be applied, when a given recording medium is chosen, in order to retain practical limits on exposure time.

C. Contrast

1. *General Considerations.* The contrast in the radiographs is related intimately to the relative roentgen-ray absorption properties and thickness of the components of the sample. Since the fractional transmitted intensity, I/I_0, through a material of thickness, x, and linear absorption coefficient, μ, is governed approximately by the relation, $I/I_0 = \exp(-\mu x)$, one finds $\partial I/I = -x\partial\mu$.

In words, the radiographic contrast between two components of the same thickness, x, but of dissimilar average linear absorption coefficients, is proportional to the product of thickness times the difference between absorption coefficients, $\Delta\mu$. In order that $\Delta\mu$ be large, the operating voltage must be low (since μ for all components, and also $\Delta\mu$ between components, increases as the roentgen-ray energy is lowered, except for absorption discontinuity effects).

The limitation on voltage is reached when I/I_0 is reduced to an excessively low value and then x must be decreased to compensate for this (as well as to increase resolution, as discussed in Section 3a above). This will lower the contrast, however. In general, both x and the operating voltage must be adjusted to obtain the maximum contrast which is compatible with high resolution and reasonable exposure times.

2. *Special Techniques.* When working with materials in the medium or high atomic number range, one can increase $\Delta\mu$ and enhance the contrast considerably by homogenizing the radiation (*e.g.*, using characteristic roentgen rays) at an energy between those energy levels corresponding to the absorption discontinuities of the elements to be separated.[6] Another method for increasing the absorption difference of biological components is to stain a particular tissue with a relatively high atomic number dye (*e.g.*, lead acetate).[7] Radiographic contrast will be discussed further in Section IV.

D. Required Materials

Considering the problems of obtaining high resolution, contrast, and reasonable exposure time, the requirements of the apparatus are as follows:

1. Ultra-fine-grain emulsion of good latitude, contrast, and sensitivity.
2. Means to provide good contact between specimen and sensitized surface.
3. Low-voltage, high-current, small projected focal spot roentgen tube.
4. Low inherent filtration.

III. ELECTRON RADIOGRAPHY

A. Technique

As described in Figure 2, electrons are ejected from a thin lead foil by roentgen radiation excited by about 250 kv. (peak); the roentgen rays are filtered to remove the soft components which might fog the film. The electrons then pass through a specimen in amounts inversely proportional to the number which are absorbed and scattered, forming a latent image on the photographic surface below.[8]

The principal advantage of electron radiography over thin specimen roentgen-ray radiography is that a conventional deep therapy medical or industrial type roent-

gen-ray unit may be substituted for the special high-current low-voltage tube described in Section II A. Furthermore, electrons do not follow the same absorption laws as do roentgen rays, and dissimilar radiographs are produced.

B. Resolution

The principal disadvantage of electron radiography is the characteristically lower resolution, by a factor of about four, obtained with respect to that of roentgen-ray radiography.

This low resolution, which permits acceptable magnifications up to only about 25 times when viewed at the normal distance, is due almost solely to the scattering of electrons in the emitter and specimen, and to the fact that electrons are emitted from the atom in nearly all directions with respect to the incident roentgen-ray beam. These electrons, ejected by about 200 kv. roentgen rays, are photoelectrons and, to a minor extent, Compton electrons. (The presence of electrons is evidenced by the fact that an intervening thin cellophane filter will reduce the film density to a small fraction of its unfiltered value.)

From an elementary theory of scattering,[9] one can calculate the average deflection due to the scattering of 1 megavolt electrons in only 25 microns of lead (the thinnest foil conveniently usable) as being more than 1 radian. The scattering angle is considerably greater for the lower electron energies encountered in electron radiography.

This is essentially why the resolution of electron radiographs is so low, and why it is so essential that the specimen be thin and in good contact with the foil and emulsion. A vacuum cassette is usually a necessity in this method of radiography.

C. Contrast

The absorption of a continuous spectrum of electrons (which is the energy distribution to be expected because of the continuous nature of the roentgen radiation) in a material follows a curve shape similar to that of roentgen-ray absorption with the important difference that a maximum electronic range exists, which is an increasing function of energy. It was found that the contrast between components is somewhat improved as the operating roentgen-ray voltage is lowered, provided the specimen is thin enough to permit adequate electron penetration. Theoretical electron range values of electrons at several energies through air, water (similar to soft biological tissue), and lead are stated in Table 1, which is an interpolation of data given by Heitler.[10] The empirical value would be on the order of 60 to 80 per cent as great since the calculated range represents an integration over a longer path due to scattering.

Table I

TOTAL PATH LENGTH OF ELECTRONS IN MILLIMETERS (THEORETICAL)

Energy	Air (NTP)	Water	Lead
100 kv.	150	0.15	0.03
200 kv.	450	0.5	0.10
500 kv.	1600	1.8	0.35

It was found, by experiment, that the best radiographs, from the standpoint of contrast, resolution, and penetration of specimen were obtained in the operating roentgen-ray range from 150 to 250 kv. (peak). The higher voltage permits good penetrability and reasonable exposure times of several minutes. The lower voltage provides good contrast with ultra-thin specimens at the expense of longer exposure times. Filtration of about 5 mm. copper plus 5 mm. aluminum (to absorb characteristic copper radiation and Compton scatter) is considered adequate. Greater filtration increases exposure times unnecessarily—lesser filtration begins to diminish contrast by increasing the background radiation density. Below 150 kv. (peak) long exposure times are required and sufficient penetration is often impossible to obtain. It is likewise impractical to operate much above 250 kv. (peak); for example, electron

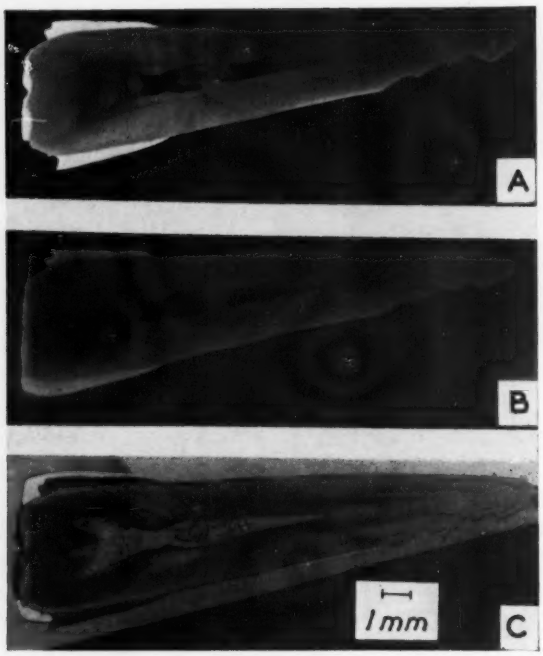

FIG. 3. Rat tooth section 25 microns thick. (*A*) Roentgen-ray radiograph. (*B*) Electron radiograph. (*C*) Photomicrograph.

radiographs taken at 1000 kv. (peak) are very diffused and show significant background density due to excessive scattering.

The Eastman 649-GH spectroscopic plates were found to provide excellent contrast, with reasonable exposures. They were used throughout the experiments, although their high resolution was not considered to be essential.

Electron radiography with beta-emitting isotopes has been used successfully.[11] No elaborate equipment is required and the background radiation density can be considerably reduced, but exposure times required are often excessively long.

IV. COMPARISONS

In Figure 3, halftone prints of a roentgen-ray radiograph, electron radiograph, and transmission photomicrograph of a section of rat tooth 25 microns thick are shown for comparison. Figure *3A* is a positive reproduction of an Eastman 649-GH plate exposed by roentgen rays emanating from a tungsten-target, 1.5 mm. projected square focal spot tube operated at 8 kv. (peak) and 380 milliampere minutes at 25 cm. from the plate. The roentgen rays were filtered by a 1 mm. beryllium window, the air, and a Wratten 87 gelatin filter. The plate reproduced in Figure *3B* was made essentially by electrons ejected from a 25 micron lead foil with roentgen rays from a tungsten-target tube, 30 cm. distant, excited at 250 kv. (peak). A sheet vinylite-covered vacuum cassette was employed, and exposure was 60 milliampere minutes with the copper-aluminum filtration described previously. Figure *3C* is a transmission photomicrograph print of the same specimen.

In Figure 4, the enlarged center portions of the first two prints are shown. Calcified tissue surrounds the dark inner regions of decalcified material. Both roentgen rays and electrons pass through the latter with ease. The three black spots seen clearly in the center of the electron radiograph are *holes* in the specimen.

Since the roentgen-ray absorption coefficient increases with about the fourth power of atomic number (except at absorption discontinuities), there is high contrast sensi-

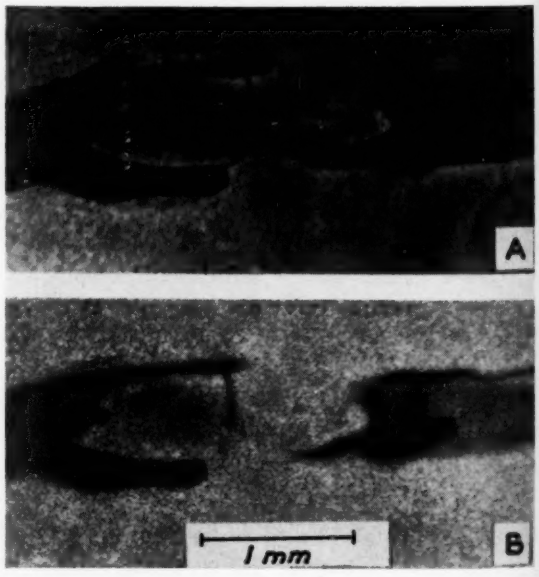

FIG. 4. Rat tooth section, center portion. (*A*) Roentgen-ray radiograph. (*B*) Electron radiograph.

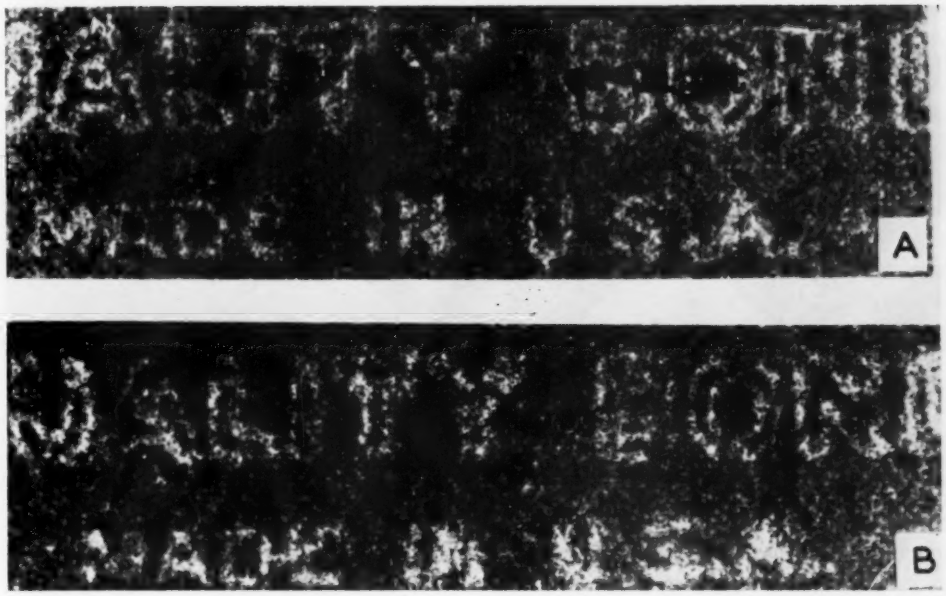

Fig. 5. Identical piece of typed bond paper taken by (*A*) visible light and (*B*) electrons.

tivity between components in the roentgen-ray radiograph which differ in *atomic number* (*e.g.*, calcified and decalcified areas) with little differentiation of physical density differences within *both* high and low atomic number regions.

The energy lost by moving electrons is directly proportional to the electron density in the matter traversed: $\rho N_0 Z/A$, where ρ is the physical density, N_0 Avogadro's number, Z atomic number, and A atomic weight. $N_0 Z/A$ is fairly constant for most elements; therefore, the electron absorption is approximately proportional to physical density. Thus, there are gradations in the electron radiograph corresponding to components differing in physical density regardless of atomic number. The contrast between the calcified and decalcified areas (large atomic number differences) in the electron radiograph reproduction is not as pronounced as in the roentgen-ray radiograph, but the density variations in the central region are sharply

Table II

Comparison of Methods

	Roentgen-Ray Radiography	Electron Radiography	Photography
Radiation medium used	5–25 kv. roentgen rays	100–200 kv. electrons	Visible light (or ultraviolet light)
Contrast obtained	Difference in atomic numbers	Difference in physical density	Difference in light transmission
Maximum permissible magnification	~100×	~25×	~2000×
Apparatus required	Low-voltage, high-current, small focal spot x-ray unit with low-absorption window (and vacuum cassette)	150–250 kv. (peak) x-ray unit (and vacuum cassette)	Photomicrographic equipment
Advantages	Simple interpretation of specimen components (without staining) with three dimensional view		High resolution

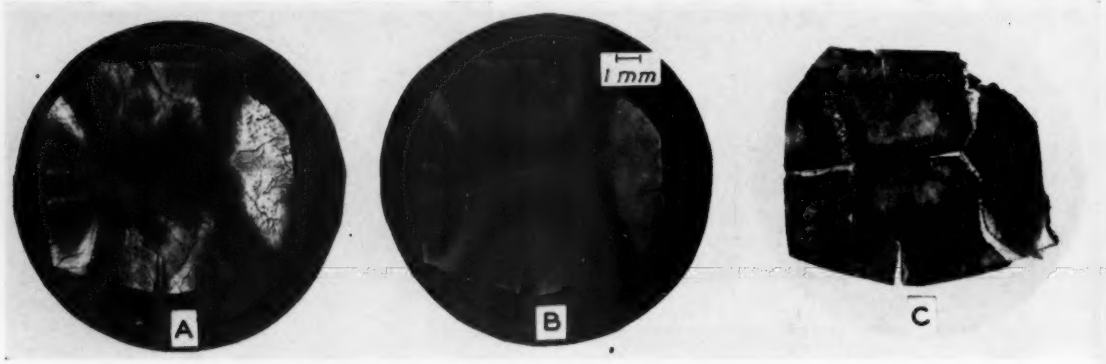

FIG. 6. Baby mouse skull 125 microns thick. (*A*) Roentgen-ray radiograph. (*B*) Electron radiograph. (*C*) Photomicrograph.

evident (note the difference between the holes and the surroundings—where holes are interpreted in terms of density changes from tissue to air).

The photomicrograph, of good resolution, reveals differences in light absorption very well but no contrast between different elements of similar light absorption properties.

The differences in the absorption characteristics of these processes are further illustrated by Figure 5, in which reproductions of an electron radiograph and a transmission photomicrograph are shown of the same piece of typed bond paper. In contrast to the previous example, the electrons, sensitive to physical density changes, do not differentiate the typing (regardless of which side of the paper faces the emulsion); the light, of course, does. A roentgen-ray radiograph, not shown, would separate the paper and typing, for the average atomic number of the typing differs somewhat from that of the paper.

Another comparison between the radiographic and photographic methods is shown in Figure 6. The specimen is a 125 micron section of a baby mouse skull which contains a bone deposition of plutonium by previous injection of a salt of the metal into the animal. The radiographs and photomicrograph are taken by the same techniques as those of Figure 4 except that the roentgen-ray radiograph was exposed at 12 kv. (peak) (the higher voltage was necessitated by the increased specimen thickness) and 40 milliampere minutes. Again, the radiographs show higher contrast between the bone and other tissue than does the photomicrograph. The electron radiograph is by far the more inferior of the reproductions because of the excessive thickness of the specimen. As previously discussed, electron scattering requires that the sample be ultra-thin. In this case, however, it is five times thicker than the rat tooth section of Figures 4 and 5.

A comparison between roentgen-ray radiography, electron radiography, and photography can best be made by reference to Table II.

Dr. Arthur Berman
Fox Project
Pratt & Whitney Aircraft
East Hartford 8, Conn.

The writers wish to thank Messrs. W. C. Herrmann and A. M. Rogers for making the photomicrographs, and Drs. W. W. Wainwright and N. P. Knowlton for providing the biological specimens.

REFERENCES

1. KIRKPATRICK, P. Approach to x-ray microscopy. *Nature*, London, 1950, *166*, 251–253.
2. ALINK, R. J., DIPPEL, C. J., and KEUNING, K. J. *Soc. Motion Picture & Television Engineering J.*, 1950, *54*, 345.
3. SHERWOOD, H. Vacuum exposure holder for microradiography. *Rev. Scient. Instruments*, 1947, *18*, 80.
4. BERMAN, A. I. Method for increasing safe power input of x-ray tubes. *Rev. Scient. Instruments*, 1950, *21*, 275–279.

5. Cosslett, V. E., and Nixon, W. C. X-Ray shadow microscope. *Nature*, London, 1951, *168*, 24.
6. Clark, G. L. Medical, biological and industrial applications of monochromatic radiography and microradiography. *Radiology*, 1947, *49*, 483–495.
7. Clark, G. L., and Bicek, E. J. In: Medical Physics. Otto Glasser, Editor. Year Book Publishers, Chicago, 1944, p. 730.
8. Trillat, J. Electronic radiography and microradiography. *J. Appl. Phys.*, 1948, *19*, 844. Berman, A. I. Electron radiography. Atomic Energy Commission Unclassified Document, AECU 1853, TID, Oak Ridge, Tenn.
9. Fermi, E. Nuclear Physics Notes. University of Chicago Press, 1950, p. 36.
10. Heitler, W. Quantum Theory of Radiation. University Press, Oxford, 1944, p. 223.
11. Westermark, T. Radiography with beta rays. *Nature*, London, 1949, *164*, 1086. Beischer, D. E. *Science*, 1950, *112*, 535.

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

Editor: LAWRENCE REYNOLDS, M.D.

Associate Editors: HARRY HAUSER, M.D., TRAIAN LEUCUTIA, M.D., RUSSELL H. MORGAN, M.D., EDWARD B. D. NEUHAUSER, M.D., WENDELL G. SCOTT, M.D.

Consulting Editorial Board: See front cover.

Published by: CHARLES C THOMAS, PUBLISHER, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued monthly. Annual subscription: United States, Mexico, Cuba, Central and South America, $15.00; Canada, $16.00; other countries, $17.00. Current single numbers, $1.50. Advertising rates submitted on application. Editorial office, 110 Professional Building, Detroit 1, Michigan. Office of publication, 301–327 East Lawrence Avenue, Springfield, Illinois. Information of interest to authors and readers will be found on page vi.

Officers and Standing Committees

AMERICAN ROENTGEN RAY SOCIETY

President: Joshua C. Dickinson, Tampa, Fla.; *President-Elect:* Paul C. Hodges, Chicago, Ill.; *1st Vice-President:* Frederic E. Templeton, Seattle, Wash.; *2nd Vice-President:* James M. Dell, Jr., Gainesville, Fla.; *Secretary:* Barton R. Young, Germantown Hospital, Philadelphia 44, Pa.; *Treasurer:* Wendell G. Scott, 35 North Central Avenue, Clayton 5, Mo.

Executive Council: J. C. Dickinson, P. C. Hodges, F. E. Templeton, J. M. Dell, Jr., B. R. Young, W. G. Scott, Lawrence Reynolds, E. L. Jenkinson, R. H. Morgan, C. A. Good, H. M. Weber, C. L. Martin, E. B. D. Neuhauser, H. O. Peterson, W. Edward Chamberlain, Chairman, 3401 N. Broad St., Philadelphia 40, Pa.

Program Committee: B. R. Young, Philadelphia, R. H. Morgan, Baltimore, Md., Lawrence Reynolds, Detroit, Mich., C. A. Good, Rochester, Minn., W. E. Chamberlain, Philadelphia, Pa., Paul C. Hodges, Chairman, 950 East 59th St., Chicago 37, Ill.

Publication Committee: H. B. Elkins, Iowa City, Iowa, R. J. Reeves, Durham, N. C., R. H. Morgan, Chairman, Baltimore, Md.

Finance Committee: R. K. Arbuckle, Oakland, Calif., J. L. Olpp, Englewood, N. J., E. L. Jenkinson, Chairman, Chicago, Ill.

Committee on Scientific Exhibits: J. F. Holt, Ann Arbor, Mich., W. A. Evans, Jr., Detroit, Mich., C. A. Good, Chairman, Rochester, Minn.

Representatives on American Board of Radiology: T. Leucutia, Detroit, Mich., B. R. Kirklin, Rochester, Minn., H. Dabney Kerr, Iowa City, Iowa.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit 1, Mich.

Fifty-sixth Annual Meeting: Palmer House, Chicago, Illinois, September 20–23, 1955.

AMERICAN RADIUM SOCIETY

President: Grant H. Beckstrand, Long Beach, Calif.; *President-Elect:* Norman A. McCormick, Windsor, Ont.; *1st Vice-President:* Herbert E. Schmitz, Chicago, Ill.; *2nd Vice-President:* Manuel Riebeling, Guadalajara, Jal, Mexico; *Secretary:* Robert E. Fricke, Mayo Clinic, Rochester, Minn.; *Treasurer:* Douglas J. Roberts, 85 Jefferson St., Hartford 6, Conn.

Executive Committee: Howard B. Hunt, Chairman, Omaha, Nebr., Edith H. Quimby, New York, N. Y., John E. Wirth, Pasadena, Calif.

Program Committee: Grant H. Beckstrand, Chairman, Long Beach, Calif., Lewis Guiss, Los Angeles, Calif., Manuel Garcia, New Orleans, La., Gray H. Twombly, New York, N. Y., Norman McCormick (ex officio), Windsor, Ont.

Publication Committee: Harry Hauser, Chairman, Cleveland, Ohio, Martin Van Herik, Rochester, Minn., J. Robert Andrews, Bethesda, Md.

Public Relations Committee: Palmer E. Wigby, Chairman, Houston, Texas, John A. Wall, Houston, Texas, James A. Chamberlin, Houston, Texas.

Janeway Lecture Committee: Douglas Quick, Chairman, New York, N. Y., Gioacchino Failla, New York, N. Y., H. Dabney Kerr, Iowa City, Iowa.

Representatives on the American Board of Radiology: Douglas Quick, New York, N. Y., B. P. Widmann, Philadelphia, Pa., Frederick W. O'Brien, Boston, Mass.

Representatives on National Committee on Radiation Protection: Edith H. Quimby, New York N. Y., Theodore P. Eberhard, Philadelphia, Pa.

Committee on Arrangements: William S. MacComb, Chairman, Houston, Texas, John A. Wall, Houston, Texas, James A. Chamberlin, Houston, Texas, Palmer E. Wigby, Houston, Texas, R. Kenneth Loeffler, Houston, Texas, Eleanor J. MacDonald, Houston, Texas, Gilbert H. Fletcher, Houston, Texas, David M. Earl, Houston, Texas, Vincent P. Collins, Houston, Texas, Grant H. Beckstrand (ex officio), Long Beach, Calif.

Thirty-eighth Annual Meeting: Shamrock Hotel, Houston, Texas, April 9–11, 1956.

EDITORIALS

FIBROUS DEFECTS IN THE CORTEX OF GROWING BONES

RADIOLUCENT areas in bone are commonly observed as incidental findings in the course of roentgenographic examination in the region of the knee in children. Radiologists have learned to recognize many as "cortical cysts" and to dismiss them from consideration as contributing to clinical symptoms. When the defects are not typical, or occur in regions other than the knee, or when local symptoms of unknown cause are present, the radiologist would like further information about the nature of the defects. This information is now available in a detailed review by Caffey[1] in a publication ordinarily not attracting the attention of radiologists.

Caffey's review is a by-product of an annual, combined roentgenologic and clinical examination of 1,000 children in communities where the effect of fluoridation of water was under study. Routine films on all children included only a single frontal projection of both knees and of one hand and forearm. The children were observed over periods of six to eight years in most cases and ranged in age from two months to nine years at the time of first examination. Detailed study of the incidence of defects in the region of the knees and their progressive changes with advancing age was possible in 154 of the 1,000 children, of whom 79 were boys and 75 girls. In no instance was any evidence found on careful clinical evaluation to suggest that the presence of a cortical defect caused abnormal local or generalized reactions in a child. Slightly more than one-third of the children showed defects; males predominated in the ratio of about 4:3. The femurs were affected approximately ten times more frequently than the tibias and sixty times more frequently than the fibulas. Unilateral defects were found as frequently as bilateral. Characteristically, defects occur on the medial and posterior aspects of the femoral shafts; only 30 of 254 defects were located lateral to the midline of the femur, and none were found on the anterior wall. No defects were observed during the first two years of life; during the third year, 7.7 per cent of the children showed defects and during the fourth year, 15.1 per cent. From the fourth through the twelfth years, the incidence ranged between 14 and 17 per cent and then declined to 10 and 8 per cent respectively, during the thirteenth and fourteenth years. At all ages, the defects were much more common in boys than in girls.

The roentgenographic changes in a series of individual defects during evolution and involution are described in detail for the first time, although variations in the appearance of different cortical defects had been documented in the second edition of Caffey's textbook,[2] and in the report of Sontag and Pyle.[3] Cortical destruction is the principal roentgenologic finding. The defects are usually oval with the long axis parallel to the axis of the shaft; they are always found in or near the ends of the shafts. The edge of the defect is sharply demarcated from the adjacent bone; there may be a zone of sclerosis, which at times is scalloped. Single and multiple lesions

[1] Caffey, J. On fibrous defects in cortical walls of growing tubular bones: Their radiologic appearance, structure, prevalence, natural course, and diagnostic significance. Advances in Pediatrics. Vol. VII. Year Book Publishers, Inc., Chicago, 1955, pp. 13–51.

[2] Caffey, J. Pediatric X-Ray Diagnosis. Second edition. Year Book Publishers, Inc., Chicago, 1950.

[3] Sontag, L. W., and Pyle, S. I. Appearance and nature of cystlike areas in distal femoral metaphyses of children. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *46*, 185–188.

may occur in the same individual. The fact had long been known that some defects maintain a constant relationship to the epiphyseal line where some believed they were generated, while others were buried progressively deeper in the shaft as the epiphysis grew away from them. Except for one report by Ponseti and Friedman,[4] the appearance and disappearance of defects had not been emphasized. Caffey describes several sequences: (1) disappearance without residual changes; (2) recurrence in the same site one or more times; (3) sclerosis and shaftward shift—rarely this is followed by expansion; (4) large defect breaks into a group of small defects which change position in relation to one another at different years of growth. The lability of the defects is a new and important observation. In addition, the first stages in the formation of a defect are reported: short segments of subperiosteal cortex are replaced irregularly by material of water density, indenting the cortex from without when the deeper layers are still intact. This results in a poorly defined, moth-eaten radiolucency and, in one case illustrated, was followed by the development of a typical defect. Lesions in bones other than those about the knee are illustrated and are considered identical in nature and significance or lack of it.

Information concerning the microscopic appearance of the defects in the group studied is not provided, but material provided by Dr. Henry L. Jaffe obtained from similar cases showed "localized, internal, fibrous hyperplasia of the periosteum with concurrent destruction of the underlying cortex." Local hemorrhage was commonly associated. Lipoid storage in connective tissue occurred in some instances as a late phenomenon. These observations are identical to those of Hatcher[5] who believed that the lesions he studied were symptomatic.

The pathogenesis of the defects is not known. Caffey postulates a traumatic origin on the basis of the age and sex incidence, the natural course of the lesions and the histologic appearance. However, he is quick to point out that the defects may be unrelated to any morbid causal factor, and concludes that until a causal agent can be demonstrated with reasonable certainty, they are best considered as normal anatomical variants.

The importance of this study and Caffey's well considered conclusions lies in the frequency with which the defects are observed in growing children and the infrequency with which they are described in standard textbooks. As an incidental observation in a healthy child, discovery without recognition may lead to mild diagnostic anxiety. Because defects must be present with equal incidence in children with various complaints, erroneous diagnoses may be favored and unnecessary procedures undertaken or necessary ones omitted as a result of the obvious "lesion" distracting attention from a less obvious morbid condition. Caffey's article contains a section on differential diagnosis including both benign and malignant conditions. The reader is referred to the original paper for these and other details.

FREDERIC N. SILVERMAN, M.D.

Children's Hospital Research Foundation
Cincinnati 29, Ohio

[4] Ponseti, I. V., and Friedman, B. Evolution of metaphyseal fibrous defects. *J. Bone & Joint Surg.*, 1949, *31-A*, 582–585.

[5] Hatcher, C. H. Pathogenesis of localized fibrous lesions in metaphyses of long bones. *Ann. Surg.*, 1945, *122*, 1016–1030.

WILLIAM H. VAN STRANDER
1874-1955

DR. WILLIAM H. VAN STRANDER was born in Edenville, New York, on January 12, 1874. He graduated from the Medical School of the University of Vermont and while an intern at the Hartford Hospital in 1900 he and Dr. Ansel Cook, a staff member of the hospital, made the first medical roentgen examination in Hartford on a nurse who had injured her knee. The exposure, according to Dr. Van Strander, required about forty minutes, and a rather severe skin reaction resulted which later necessitated a skin graft.

Dr. Van Strander opened an office in

Hartford in 1902 with a static machine, and in 1903 he added a 12 inch coil. He was the first physician in Hartford to operate a roentgen-ray machine to any extent for the benefit of other physicians in the city. He claimed to have done the first gastro-intestinal roentgen examination in Connecticut between 1903 and 1904 when he diagnosed a duodenal ulcer, later confirmed by operation at the Mayo Clinic.

A Roentgen-Ray Department was installed at St. Francis Hospital in Hartford in 1905 and the annual report of that hospital for 1907 lists Dr. Van Strander as the radiologist. He was associated with St. Francis Hospital for approximately thirty-five years in that capacity. He maintained his private office until less than one year from his death on February 13, 1955, at the age of eighty-one after more than a half century of continued interest in radiology. He is survived by his wife, Mrs. Helen Van Strander, and two sons, a daughter, and four grandchildren.

WENDELL C. HALL, M.D.

LOUIS EDEIKEN
1894-1955

THE death on April 11, 1955, of Dr. Louis Edeiken at the age of sixty-one, took from the ranks of second generation American Radiologists one of its most able and dedicated members.

Dr. Edeiken was connected with the Albert Einstein Medical Center and formerly the Mt. Sinai Hospital of Philadelphia for more than thirty-five years where he was an attending Chief of Radiology since 1933. Never during this long hospital association did Dr. Edeiken relinquish his own private practice of radiology in central city offices where, latterly, he was joined by his son, Dr. Jack Edeiken. This independence of hospital control over his private practice was always a heartening example to those who so frequently became entangled in hospital-radiologist relationships.

A graduate of the Medical-Chirurgical College of Philadelphia in 1914, blindness of the right eye prevented him from assuming military duties with the Armed Forces during the First World War. Nevertheless, he actively participated in strenuous military home-front assignments. Likewise, during the Second World War, he was an active member of a Medical Advisory Board for Selective Service.

In spite of the demands on his time by hospital and private practice, he enthusiastically took part in various teaching programs and in clinical research. He was Clinical Professor of Radiology at Hahnemann Medical College since 1938 and an Associate in Radiology in the Graduate School of Medicine, University of Pennsylvania since 1941.

Among his numerous publications, those concerned with scleroderma and sterility stand out most prominently.

Family and friends were Louis Edeiken's dearest treasures. Blessed with an abundance of both, never was he more happy than when he was "doing" for them. His selflessness and dedication to others found their greatest expression in his home and in his fraternal life. As a member, then president and chairman of the Board of Trustees of Phi Lambda Kappa Medical Society over a period of twenty-nine years, he was its most illustrious representative in fraternal matters national or international. Gentle as he was, this was never misinterpreted as weakness. Time and again his ability to overcome obstacles by reasoning, gentle persuasion and, when necessary, firmness made him a valued addition to any group. In his memory the Phi Lambda Kappa Medical Society has created the Louis Edeiken Medical Student Loan Fund and Lectureship. Always attentive to nu-

merous social, religious, and civic activities his interests were also sparked in the burgeoning of the new medical school of Israel.

Dr. Edeiken was a Fellow of the American College of Radiology, a Diplomate of the American Board of Radiology, a member of the American Medical Association, a member of the American Roentgen Ray Society, and the Philadelphia Roentgen Ray Society.

Among his survivors are his devoted wife, Dr. Nettie S. Edeiken, his two sons, Dr. Jack and Dr. Stanley Edeiken, a brother Dr. Joseph Edeiken and two daughters, Mrs. Blanche Kramer and Miss Janet Edeiken.

To a wide host of friends, his sincere loving kindness will always be remembered and cherished, an abiding tribute to a beloved colleague.

J. Gershon-Cohen, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting: Palmer House, Chicago, Ill., Sept. 20–23, 1955.

AMERICAN RADIUM SOCIETY
Secretary, Dr. R. E. Fricke, Mayo Clinic, Rochester, Minn. Annual meeting: Shamrock Hotel, Houston, Texas, April 9–11, 1956.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill., Dec. 11–16, 1955.

AMERICAN COLLEGE OF RADIOLOGY
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: 1956, to be announced.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual Meeting: 1956, to be announced.

EIGHTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary General, Dr. José Noriega, Calle del Oro 15, Mexico 7, D. F. Meets Mexico City, July 22–28, 1956.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. J. J. Riordan, 550 W. Thomas Rd., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Norton, 843 Donaghey Bldg., Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Rayle, Jr., 478 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. Daniel G. Butera, 709 E. 39th St., Brooklyn, N. Y. Meets first Thursday of each month October through April.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, October through April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Clayton G. Weig, 135 Linwood Ave., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse, N. Y. Meets January, May, November.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. F. A. Riebel, 15 West Goodale St., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Columbus Athletic Club, Columbus, Ohio.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. E. Burns Lewis, 670 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month, October to April inclusive at the Sheraton Hotel.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. A. S. Tucker, 2020 East 93 St., Cleveland 6, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. S. A. Patterson, Larimer County Hospital, Fort Collins, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. T. J. Crowe, 53 Center St., Northampton, Mass. Meets second Friday October and April.

DALLAS-FORT WORTH RADIOLOGICAL CLUB
Secretary, Dr. A. H. Keene, 3707 Gaston Ave., Dallas, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. F. Lang, Harper Hospital, Detroit, Mich. Meets monthly first Thursday, Oct. through May, at David Whitney House, 4421 Woodward Ave., at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. D. H. Gahagen, 320 Sweet Bldg., Fort Lauderdale, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. B. H. Malone, 1406 Reynolds St., Brunswick, Ga.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary, Dr. A. S. Capi, 300 N. 20th Ave., Hollywood, Fla. Meets monthly third Wednesday at 8 P.M. at various South Florida Hospitals.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Owsley, 6409 Fannin, Houston 25, Texas. Meets fourth Monday each month at Texas Children's Hospital.

IDAHO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred M. Stone, St. Lukes Hospital, Boise, Idaho. Meets annually at same time as Idaho State Medical Association.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. C. A. Stayton, 313 Hume-Mansur Bldg., Indianapolis 4. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society, The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. A. M. Cherner, Hays, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. David Shapiro, 217 East Chestnut St., Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Kings County Radiological Society
Secretary, Dr. Solomon Maranov, 1450 51st St., Brooklyn 19, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Oscar Harvey, 3741 Stocker St., Los Angeles 8, Calif. Meets second Wednesday each month at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. W. A. Russell, Augusta General Hospital, Augusta, Maine. Meets in June, October, December and April.

Maryland Radiological Society
Secretary, Dr. Paul W. Roman, 1810 Eutaw Place, Baltimore 17, Maryland.

Memphis Roentgen Society
Secretary, Dr. B. E. Greenberg, 294 Annella St., Memphis 11, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. R. W. Byrne, 3321 N. Maryland Ave., Milwaukee 1, Wis. Meets monthly on fourth Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. John R. Hodgson, Mayo Clinic, Rochester, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

Mississippi Radiological Society
Secretary, Dr. James M. Packer, 621 High St., Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. Grant P. Raitt, Suite 413 Medical Arts Bldg., Billings, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Alan E. Baum, Hicksville, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Stanley M. Wyman, Massachusetts General Hospital, Boston, Mass. Meets third Friday of each month, October through May, at Hotel Commander, Cambridge, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. M. H. Poppel, 303 E. 20th St., New York 3, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. W. C. Sternbergh, Charlotte Memorial Hospital, Charlotte, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. Marianne Wallis, St. Joseph's Hospital, Minot, N. Dak. Meetings by announcement.

North Florida Radiological Society
Secretary, Dr. Ivan Isaacs, 1645 San Marco Blvd., Jacksonville, Florida. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. D. H. Baxter, Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesdays of October, November March and April.

Northern California Radiological Society
Secretary, Dr. H. B. Stewart, 2920 Capitol Ave., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. M. M. Thompson, Jr., 319 Toledo Medical Bldg., Toledo 2, Ohio. Annual meeting, Mayflower Hotel, Akron, Ohio, May 20–22, 1955.

Oklahoma State Radiological Society
Secretary, Dr. John R. Danstrom, Medical Arts Bldg., Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. F. C. Shipps, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. R. Raines, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. H. M. Stauffer, Temple University Hospital, Philadelphia 40, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. N. Tannehill, 500 Penn Ave., Pittsburgh 22, Pa. Meets second Wednesday of month, October through June at Hotel Roosevelt.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Paul W. Roman, 1810 Eutaw Place, Baltimore 17, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of Greater Cincinnati
Secretary, Dr. R. J. Neubauer, 831 Carew Tower, Cincinnati, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. George W. Henry, 1133 Punchbowl, Honolulu, T. H. Meets third Monday evening of each month at designated hospitals.

Radiological Society of Greater Kansas City
Secretary, Dr. E. H. Stratemeier, Jr., 1010 Rialto Building, Kansas City 6, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. W. S. Neal, 602 Pere Marquette Bldg., New Orleans, La. Meets annually during Louisiana State Medical Society Meeting.

Radiological Society of New Jersey
Secretary, Dr. G. G. Green, 601 Grand Ave., Asbury Park, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

Radiological Society of Southern California
Secretary, Dr. J. B. Irwin, 1831 Fourth Ave., San Diego, Calif.

Richmond County Radiological Society
Secretary Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Henry H. Forsyth, Jr., 40 Meigs St., Roch-

ester, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. John H. Freed, 4200 East Ninth St., Denver 7, Colo. Mid-Summer Conference, Shirley-Savoy Hotel, Denver, Colo., Aug. 18–20, 1955.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. Edwin C. Ernst, Jr., 3720 Washington Ave., St. Louis 8, Mo. Meets fourth Wednesday each month, except June, July, Aug. and Sept.

SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas Meets Brooke Army Medical Center Officers Mess first Monday of each month.

SAN DIEGO RADIOLOGICAL SOCIETY
Secretary, Dr. J. C. Asher, 2001 Fourth Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. T. M. Fullenlove, 110 El Verano Way, San Francisco 27, Calif. Meets quarterly at Grison's Steak House.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. H. R. Morris, 1027 "D" St., San Bernardino, Calif.

SECTION ON RADIOLOGY, CONNECTICUT STATE MEDICAL SOCIETY
Secretary, Dr. J. Burbank, Meriden Hospital, Meriden, Conn. Meetings are held bi-monthly.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary, Dr. F. V. Schumacher, 1835 Eye St., N.W., Washington 6, D. C. Meets Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. George E. Irwin, Jr., 703 N. East St., Bloomington, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

SOUTH BAY RADIOLOGICAL SOCIETY
Secretary, Dr. T. N. Foster, 630 E. Santa Clara St., San Jose, Calif. Meets second Wednesday of each month.

SOUTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. S. W. Lippincott, 103 Rutledge Ave., Charleston, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. G. K. Henshall, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next Meeting, Fort Worth, Texas, Jan. 20–21, 1956.

TRI-STATE RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Hendershot, 118 S.E. First St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

UPPER PENINSULA RADIOLOGICAL SOCIETY
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

UTAH STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Eva L. Gilbertson, 1317 Marion St., Seattle 4, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

WEST VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. W. Paul Elkin, 515 Medical Arts Bldg., Charleston, W. Va. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER RADIOLOGICAL SOCIETY
Secretary, Dr. M. G. Priestman, New Rochelle Hospital, New Rochelle, N. Y. Meets on third Tuesday of January and October and on two other dates.

WISCONSIN RADIOLOGICAL SOCIETY
Secretary, Dr. W. W. Moir, 309 Geele Ave., Sheboygan, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. T. English, 2000 Van Ness Ave., San Francisco 9, Calif. Meets monthly, third Thursday at 7:45 P.M., Lane Hall, Stanford University Hospital.

CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

SOCIEDAD MEXICANA DE RADIOLOGÍA, A. C.
Calle del Oro No. 15, Mexico 7, D. F.
Secretary General, Dr. Guillermo Santín. Meets first Monday of each month.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
Secretary, Dr. Rafael A. Blanes, P. O. Box 9067, Santurce, Puerto Rico.

SOCIEDAD RADIOLÓGICA PANAMEÑA
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86, Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

BRITISH EMPIRE

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
Secretary, Dr. Jean-Louis Léger, 1560 Sherbrooke St., Montreal, Que. Meets four times a year.

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1

FACULTY OF RADIOLOGISTS
Honorary Secretary, Dr. R. A. Kemp Harper, 45, Lincoln's Inn Fields, London, W.C.2, England.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary-Treasurer, Dr. D. L. McRae, Suite 305, 1555 Summerhill Avenue, Montreal 25, Que. Meetings January and June.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALES
General Secretary, Dr. Ls Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

COLLEGE OF RADIOLOGISTS (AUSTRALIA AND NEW ZEALAND)
Honorary Secretary, Dr. Alan R. Colwell, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W.

South America

Asociacion Argentina de Radiologia
Secretary, Dr. Lidio G. Mocsa, Avda. Gral. Paz 151 Córdoba, Argentina. Meetings held monthly.

Sociedad Argentina de Radiologia, Junta Central, Buenos Aires
Secretary, Dr. Oscar F. Noguera, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Argentina de Radiologia, Filial del Litoral, Sede, Rosario
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 19 ha 663 Italia St., Rosario, Santa Fe.

Sociedad Argentina de Radiologia, Filial del Sur, Sede, La Plata
President, Prof. Dr. Jose P. Uslenghi, La Plata. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Centro Sede, Córdoba
President, Prof. Dr. David L. Caro, Corro 86, Córdoba. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Norte, Sede, Tucuman
President, Dr. Julio Mendez Valladares, Rivadavia 358, Tucuman. Meets monthly.

Sociedad Argentina de Radiologia, Filial de Cuyo Sede, Mendoza.
President, Dr. J. A. Orfila, Hospital Central, Mendoza. Meets monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Colombiana de Radiologia
Secretary, Dr. Luis E. Lozano B., Carrera 12 No. 20–69, 6° piso, Bogota, Colombia. Meets last Thursday of each month.

Sociedad Peruana de Radiologia
Secretary General, Dr. Amador Holgado V. Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary General, Dr. Roberto Francois, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Sociedad Venezolana de Radiología
Secretary, Dr. Otto Paz, Centro Médico, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Austrian Roentgen Society
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Société Française d'Electro-Radiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord
Secretary-General, Dr. A. Dariaux, 9 bis, Boulevard Rochechouart, Paris IX°, France.

Ceskoslovensk společnost pro rëntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgen-Gesellschaft
Secretary, Dr. med. Peter Kröker, Evangelisches Krankenhaus, Huyssens Stiftung, (22a) Essen.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

Nederlandse Vereniging voor Electrologie en Roentgenologie
Secretary, Dr. B. J. Rethmeier, Bornsestraat 378, Almelo, Netherlands.

Polish Society of Radiology
Secretary, Dr. Juljusz Borejko, 59 Nowogrodzka St., Warsaw, Poland.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10:30, X-Ray Dept., Akademia Gdansk.

Wroclaw Section, Polish Society of Radiology
Secretary, Dr. R. Marciniak, Akad. Medyczna Wroclaw, 68 Curie-Sklodowska St., Wroclaw, Poland. Meets first Sunday of each month, X-Ray Department, Akademia Medyczna Wroclaw.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S.I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société-Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The list of Meetings of Radiological Societies is published in every other issue of the Journal.

ARKANSAS RADIOLOGICAL SOCIETY

The Arkansas Radiological Society held its annual breakfast meeting at the Arlington Hotel, Hot Springs, on May 30, 1955. The following officers were elected for 1955–1956: *President*, Dr. George Burton; *Vice President*, Dr. Ed Gray; *Secretary-Treasurer*, Dr. Joe Norton, 843 Donaghey Bldg., Little Rock, Ark.

MAINE RADIOLOGICAL SOCIETY

At the recent annual meeting of the Maine Radiological Society, the following officers were elected for the coming year: *President*, Dr. G. E. Clifford Logan, Portland, Maine; *Vice President*, Dr. Hugh A. Smith, Bangor, Maine; *Secretary-Treasurer*, Dr. Walter A. Russell, Augusta General Hospital, Augusta, Maine.

NEW ENGLAND ROENTGEN RAY SOCIETY

At the annual meeting of the New England Roentgen Ray Society the following officers were elected for the year 1955–1956: *President*, Dr. Max Ritvo; *Vice President*, Dr. A. B. Soule, Jr.; *Treasurer*, Dr. Magnus I. Smedal; *Secretary*, Dr. Raymond A. Dillon, 24 Wedgemere Ave., Winchester, Massachusetts. Meetings are held in the evening on the third Friday of each month.

NEW YORK ROENTGEN SOCIETY

The new officers of the New York Roentgen Society recently elected for 1955–1956 are as follows: *President*, Dr. Jacob Fried; *Vice President*, Dr. Sidney Rubenfeld; *Secretary*, Dr. Maxwell H. Poppel, 303 E. 20th St., New York 3, New York; *Treasurer*, Dr. John A. Evans.

PITTSBURGH ROENTGEN SOCIETY

At the meeting of the Pittsburgh Roentgen Society, held on June 8, 1955, the following officers were elected for the coming year: *President*, Dr. Joseph E. Malia; *Vice President*, Dr. Erwin Beck; *Secretary*, Dr. Norman Tannehill, 500 Penn Ave., Pittsburgh 22, Pennsylvania; *Treasurer*, Dr. Harrison Richardson. The regular meetings are held on the second Wednesday of the month, October through June.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI

At a recent meeting of the Radiological Society of Greater Cincinnati, the following officers were elected for 1955–1956: *President*, Dr. William R. Dickens; *Vice President*, Dr. Benjamin Schneider; *Secretary-Treasurer*, Dr. Richard J. Neubauer, 831 Carew Tower, Cincinnati, Ohio.

RADIOLOGICAL SOCIETY OF HAWAII

At the June meeting of the Radiological Society of Hawaii the following officers were elected for the coming year: *President*, Dr. Philip S. Arthur; *Vice President*, Dr. Hong Chong Chang; *Secretary-Treasurer*, Dr. George W. Henry, 1133 Punchbowl, Honolulu, T. H. The Society meets on the third Monday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA

In May, 1955, the Radiological Society of Louisiana held its annual meeting and the following officers were elected for the coming year: *President*, Dr. G. M. Riley, Shreveport, Louisiana; *Vice President*, Dr. Manuel Garcia, New Orleans, Louisiana; *Secretary-Treasurer*, Dr. W. S. Neal, 602 Pere Marquette Bldg., New Orleans, Louisiana.

TENNESSEE RADIOLOGICAL SOCIETY

The newly elected officers of the Tennessee Radiological Society are: *President*, Dr. John M. Wilson, Memphis, Tennessee; *Vice President*, Dr. Ben R. Mayes, Nashville, Tennessee; *Secretary-Treasurer*, Dr. George K. Henshall, 311 Medical Arts Bldg., Chattanooga 3, Tennessee. The meeting at which the officers were elected was held in conjunction with the State Medical Association Meeting in Chattanooga, Tennessee on April 11, 1955. Dr. Manuel Garcia was guest speaker and discussed "Treatment of Carcinoma of the Uterine Cervix."

UPPER PENINSULA RADIOLOGICAL SOCIETY

At the recent meeting of the Upper Peninsula Medical Society a new group was formed consisting of the radiologists of the Upper Peninsula of Michigan. The group will be known as the Upper Peninsula Radiological Society. The following officers were elected: *President*, Dr. L. Grant Glickman, Menominee, Michigan; *Vice President*, Dr. T. Boyd Bolith, Marquette, Michigan; *Secretary*, Dr. Arthur Gonty, Menominee, Michigan. The group will meet quarterly.

RESIDENCY TRAINING IN RADIOLOGY

In collaboration with the Joint Residency Review Committee for Radiology, representing the American Board of Radiology and the Council on Medical Education and Hospitals of the American Medical Association, the American Board of Radiology has adopted the following policy:

> Effective immediately, hospitals and institutions making initial application for approval for residency training in radiology must qualify for full three-year approval. No new residencies of one or two years' duration will be approved unless they are integrated with or contributory to a fully approved program.
>
> It is strongly recommended that all hospitals and institutions presently approved for residency training in radiology develop programs offering complete training, either intramurally or through affiliation by June 30, 1957.

The Joint Residency Review Committee has been established to act in behalf of these two organizations in approving residency programs in this specialty. The present policy relating to future approval for full training only has been adopted to insure that residents receive integrated training with graded responsibility and continuity of supervision during their three years' residency. The requirement of a fully organized program in qualifying for approval is in accord with sound educational principles as adopted in several other specialty fields. The Committee does not believe it to be in the best interest of the resident that he be permitted to take a year or more of isolated training in two or three unrelated programs in qualifying for Board examination.

Residents who have acepted appointment to programs which are presently approved for less than three years will receive full credit for their training on the basis of the residency's present status, through June 30, 1957.

Sincerely yours,
B. R. Kirklin, M.D.
Secretary-Treasurer
American Board of Radiology

CONTINUATION COURSE IN RADIOLOGY

The University of Minnesota will present its annual Continuation Course in Radiology for Radiologists at the Center for Continuation Study November 7 to 12, 1955. The subject of this year's program will be neuroradiology.

The guest faculty will include Drs. Donald L. McRae, Chief of Radiology, Montreal Neurological Institute, McGill University Faculty of Medicine, Montreal; Ernest H. Wood, Jr., Professor of Radiology, University of North Carolina School of Medicine, Chapel Hill; and Professor Erik Lindgren, Professor of Radiology, Serafimerlasarettet, Stockholm, Sweden.

The program will be presented under the direction of Dr. Leo G. Rigler, Professor and Head, Department of Radiology, and Dr. H. O. Peterson, Clinical Professor, Department of Radiology, University of Minnesota Medical School.

Lodging and meal accommodations are available at the Center for Continuation Study.

GRANTS IN AID FOR TRAINING COURSES IN RADIOISOTOPE FIELD

Realizing the need for increased training and educational opportunities in the radioisotope field, Abbott Laboratories has set aside the sum of $2,000, from which grants in aid will be made to suitable training courses or programs offered by recognized educational institutions and hospitals.

Instruction must be of such a nature to provide in the course, or as an adjunct thereto, the basic information and clinical experience required by the Atomic Energy Commission for the use of at least one isotope.

Individual grants may be expended for the improvement of laboratory facilities, for the procurement of guest speakers, or in any other way the directors of the training programs may elect.

D. L. Tabern
Head, Department of
Radioactive Pharmaceuticals
Abbott Laboratories

CONGRESS OF NEUROLOGICAL SURGEONS

The Congress of Neurological Surgeons will hold its meeting at the Statler Hotel, Los Angeles, California, October 27, 28, and 29, 1955. The Secretary of the Congress is Dr. Philip D. Gordy, 1007 Delaware Avenue, Wilmington, Delaware.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Neck and Chest

Haas, L. L., and Baker, B.: Neck roentgenograms in the diagnosis of esophageal carcinoma 540

Haas, L. L., and Baker, B.: Tumor outline of esophageal carcinoma 540

Gershon-Cohen, J., and Ingleby, Helen: Neglected roentgenography of breast disease 540

Giraud, G., *et al.:* Pneumolaminagraphy 540

Stecken, A.: On varices of the lung 541

Gelli, G.: Pulmonary mycosis (moniliasis) in cavitary form; with particular reference to the surgical treatment 541

DeBord, R. A., and Sibilsky, C. E.: Congenital obstructive emphysema treated by lobectomy 541

Keats, T. E., and Crane, J. F.: Cystic changes of the lungs in histiocytosis 542

Lucherini, T.: Articular and gynecomastic manifestations in hypertrophic pulmonary osteoarthropathy; study of the problem of pathologic etiology 542

Boucot, Katharine R., *et al.:* Philadelphia pulmonary neoplasm research project; preliminary report 542

Lemmon, M. L., Kirklin, J. W., and Dockerty, M. B.: Intralobar bronchopulmonary sequestration studied by multicolored vinyl acetate cast; report of case 543

Lalli, A., Carlson, R. F., and Adams, W. E.: Intralobar pulmonary sequestration; report of three cases, with two additional cases of agenesis of the right upper and middle lobes in combination with anomalous systemic vessels 543

Ciarpaglini, L., and Fusi, G.: Considerations on the possibilities and the advantages of laminagraphy compared to bronchography in the diagnosis of lung tumors 543

Siegal, W., Plunkett, R. E., and Locke, B. Z.: Case findings from routine chest roentgenograms; mass surveys of communities versus general hospital admissions 543

Kremens, V.: Demonstration of the pericardial shadow on the routine chest roentgenogram: a new roentgen finding; preliminary report. 544

Haight, C. Sloan, H., and Rubel, W. F.: Coarctation of the aorta 544

Abdomen

Bulgrin, J. G., and Holmes, F. H.: Eventration of the diaphragm with high renal ectopia; a case report 545

Hillemand, P., and Barré, Y.: On the etiology of certain diaphragmatic hernias through the esophageal hiatus 545

O'Connor, F. J., and Ritvo, M.: Diagnosis of hiatus hernia on plain roentgenograms of thorax and abdomen 545

Whitehouse, W. M.: Correlation of surgical pathology with telepaque cholecystography in doses of two grams 545

Gaylis, H., and Gunn, K.: Transduodenal cholangiography 546

Teschendorf, W.: Visualization of the biliary tract by means of biligrafin, especially after cholecystectomy 546

Anacker, H.: The filling and emptying functions in the bile ducts 546

Lange, L.: Simultaneous cholecysto-cholangiography; preliminary report 547

Rosenbaum, F.-J.: The laparoscopic cholangiography 547

Twiss, J. R., *et al.:* Postcholecystectomy oral cholangiography 547

Davis, L. A., Knoefel, P. K., and Pirkey, E. L.: Factors influencing the roentgen visualization of the gastric mucosa 547

Friedlander, F. C., and Kushlick, P.: Trichobezoar 548

Ottoman, R. E., and Woodruff, J. H., Jr.: Polypoid diseases of the stomach 548

Ludin, H.: Combined pneumography of the pancreas 549

Wilson, J. W.: The diagnosis of abdominal cysts in infants and children 549

Root, G. T., Christensen, B. H., and Peterson, C. A.: Intussusception: an eight year survey 550

Steinbach, H. L., *et al.:* The potential hazard of enemas in patients with Hirschsprung's disease 550

Genitourinary System

Hope, J. W., and Campoy, F.: The use of carbonated beverages in pediatric excretory urography 550

Moore, C. A., and Dodson, C. C.: Urinary

tract calculi in children; renal and vesical calculi in an eight-month-old child..... 551
Baron, G. J., and Koenemann, R. H.: Arteriovenous fistula of the renal vessels; a case report..... 551

Nervous System

Breit, A., and Wiedenmann, O.: Results of gas myelography..... 551
Brierre, J. T., and Colclough, J. A.: Total myelography; complete visualization of the spinal subarachnoid space..... 552

Skeletal System

McCormack, L. J., Dickson, J. A., and Reich, A. R.: Actinomycosis of the humerus..... 552
Smith, A. McE.: Sprain of the pisiform-triquetral joint..... 552
Mason, M. L.: Traumatic dislocation of the hip in childhood..... 553
McCallum, R. I., *et al.:* Avascular necrosis of the femoral heads in a compressed air worker..... 553
Lyford, J., III: The fate of femora with pathologic fractures due to malignant tumors, stabilized by intramedullary nails, as shown by serial roentgenograms..... 553
Ravelli, A.: The roentgen appearance of the proximal third of the tibia..... 554
Schwabe, H. K.: Tuberculous changes of the band apparatus of the spine; a contribution to the mode of infection of tuberculous spondylitis in adults..... 554
Walker, C. S.: Calcification of intervertebral discs in children..... 554
Van de Berg, F., and Crèvecoeur, M.: Lumbar disco-radiculography by means of monoiodine hydrosoluble contrast medium. 554
Kloss, K.: Treatment of the complications of myelography following use of water soluble contrast media..... 555
Lamb, D. W.: Localized osteochondritis of the lumbar spine..... 555
Odell, R. T., and Key, J. A.: Lumbar disk syndrome caused by malignant tumors of bone..... 556
Williams, R.: Complete protrusion of a calcified nucleus pulposus in the thoracic spine.. 556
Dahlin, D. C., *et al.:* Aneurysmal bone cysts. 556
Kellsey, D. C., and Sproat, H. F.: Echinococcus disease of bone; report of a case.... 557
Bierling, G., and Reisch, D.: The Sudeck syndrome after fractures..... 558
Liess, G.: Multiple symmetrical repair zones (Milkman's syndrome) of uncommon etiology and localization..... 558
Singer, M.: Fanconi's syndrome; treatment by massive dosage of calciferol and two-stage osteotomy..... 559
Gersten, A., and Lecomte-Ramioul, S.: Roentgenological study of the costochondral junctions in rickets..... 560
Dechaume, M., *et al.:* Concerning an observation of the Albright syndrome..... 560
Cameron, J. A. P., Young, W. B., and Sissons, H. A.: Metaphysial dysostosis.... 560
Jackson, W. P. U., *et al.:* Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. I. Familial metaphyseal dysplasia and craniometaphyseal dysplasia; their relation to leontiasis ossea and osteopetrosis; disorders of "bone remodeling"..... 561
Jackson, W. P. U., Hanelin, J., and Albright, F.: Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. II. Multiple epiphyseal dysplasia; its relation to other disorders of epiphyseal development..... 562
Jackson, W. P. U., Hanelin, J., and Albright, F.: Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. III. Progressive diaphyseal dysplasia..... 562
De Pape, A. J.: Multiple pseudo-cystic tuberculosis of bone..... 563
Potter, C. M. C.: Osteomyelitis of the newborn..... 563
Snapper, I., *et al.:* Osteomalacia in New York. 564
Engfeldt, B., Engström, A., and Zetterström, R.: Biophysical studies of the bone tissue in osteogenesis imperfecta..... 564
Janes, J. M., Higgins, G. M., and Herrick, J. F.: Beryllium-induced osteogenic sarcoma in rabbits..... 564

Blood and Lymph System

Fiandra, O., *et al.:* Nonobstructive dilatation of the jugular vein..... 565
Petrie, E. A.: Aberrant right subclavian artery..... 565
Szilagyi, D. E., and Overhulse, P. R.: Segmental aorto-iliac and femoral arterial occlusion; treatment by resection and arterial graft replacement..... 566
Paden, P. A., and Matz, F. A., Jr.: Benign systemic hemangiomatosis without cutaneous involvement; a confusing diagnostic problem..... 566
Drucker, V., and Rodriguez, C. E.: Extensive bilateral calcification within adrenal hemorrhage..... 566

ROENTGEN DIAGNOSIS

NECK AND CHEST

HAAS, LEWIS L., and BAKER, BERNARD. Neck roentgenograms in the diagnosis of esophageal carcinoma. *Radiology*, Feb., 1955, *64*, 234–240.

Esophageal carcinoma is usually diagnosed roentgenographically by analysis of the intraluminal defects as shown in the barium-filled esophagus. The direct roentgen signs, which are so frequently visible, are often neglected. For lesions of the cervical and upper thoracic esophagus, sagittal and lateral roentgenograms are a valuable diagnostic aid.

Among the direct roentgen signs are deformities of the pyriform sinuses and the hypopharynx and widening of the prevertebral soft tissue shadow. The prevertebral widening does not represent a direct delineation of the tumor, but is attributable to both the space-occupying lesion and the secondary mechanical distention. The widened space is characteristically wedge shaped. Other space-occupying lesions and some inflammatory alterations arising from the vertebral bodies, hypopharynx and mediastinum may produce widening of the prevertebral spaces. The widening in these instances, however, is usually not wedge shaped.—*A. W. Sommer, M.D.*

HAAS, LEWIS L., and BAKER, BERNARD. Tumor outline of esophageal carcinoma. *Radiology*, Feb., 1955, *64*, 241–248.

The roentgen diagnosis of carcinoma of the esophagus is usually based upon the alterations seen in the barium-filled lumen. Very commonly little attention is paid to the external soft tissue shadow, which represents the intramural and/or extraesophageal extension of the tumor. Careful study of this soft tissue shadow of the tumor can yield valuable information regarding the length, width, and depth of the lesion. Visualization of tumor in the upper thoracic segment of the esophagus is aided by the preesophageal location of the trachea, which offers a sharp contrast to the tumor tissue density. Tumors below the carina and in the subdiaphragmatic segment of the esophagus are less readily identified by their soft tissue shadow.

Any technic resulting in high-contrast films should permit visualization of the tumor shadow in most instances. Information gained in this manner is valuable from a prognostic as well as therapeutic standpoint.—*A. W. Sommer, M.D.*

GERSHON-COHEN, JACOB, and INGLEBY, HELEN. Neglected roentgenography of breast disease. *J.A.M.A.*, Jan. 22, 1955, *157*, 325–326.

In the authors' series of 102 consecutive carcinomas of the breast 85 (83 per cent) were diagnosed correctly and 14 more (14 per cent) were suspected from the roentgenographic findings. From clinical studies only 55 (54 per cent) were diagnosed correctly while 26 more (25 per cent) were suspected. The remaining 21 lesions (21 per cent) were labeled benign by the clinician while a similar error was made in only 3 cases (3 per cent) by the radiologist. In a series of 316 benign tumors, 287 (91 per cent) were diganosed correctly and 29 (79 per cent) were suspected from roentgenographic studies. There were no incorrect diagnoses. From clinical studies, the correct diagnosis was made in 225 cases (71 per cent) and there were 79 suspicious cases (25 per cent). An incorrect diagnosis was made in 12 cases (14 per cent).

The authors conclude that a higher accuracy in differentiating benign from malignant lesions can be obtained by roentgenography than by clinical examination alone. Periodic roentgenographic studies of normal breasts could be used effectively to detect and care for carcinoma earlier than is now usually done in patients who accidentally discover the lesions.—*James J. McCort, M.D.*

GIRAUD, G., BÉTOULIÈRES, P., LATOUR, H., and PÉLISSIER, M. La pneumostratigraphie. (Pneumolaminagraphy.) *Presse méd.*, Dec. 25, 1954, *62*, 1781–1785.

The term pneumolaminagraphy applies to body section roentgenography following retroperitoneal air insufflation. The air which surrounds the organs and structures of the abdomen and mediastinum provides the necessary contrast for their visualization.

Air filtered through cotton-wool is injected into the precoccygeal space within fifteen to twenty minutes. The injection is usually harmless; only 1 fatal accident in 500 patients examined was reported. Injection into the retropubic space is used in patients where the knee-chest position is not well tolerated. The retroxiphoid injection is used for examination of the anterior mediastinum. The transtracheal injection is carried out in the subglottic space and by air infiltration of the tracheo-eosophageal space a good visualization of the mediastinum is obtained. The retromanubrial air injection is used for visualization of the thymus.

The laminagrams are best obtained about ten to twelve hours after air injection when diffusion of the air is complete. Sections are taken with the patient upright in the anteroposterior, lateral and oblique projections. Horizontal laminagrams are valuable in certain cases.

In the following mediastinal diseases the method was found useful: (1) Esophageal lesions. Here the external contours of the organ, adhesions and enlargement of para-esophageal lymph nodes were clearly demonstrated. (2) Aortic aneurysms. Here accompanying mediastinitis was well visualized. (3) In coarctation of the aorta the narrowed segment, poststrictural dilatation and collateral circulation

were shown. (4) Lesions of the pulmonary arteries. (5) Pathology of the mediastinal venous system. (6) Anomalies of the heart chambers. (7) The external contours of the diseased trachea and main bronchi as well as the mediastinal pleura. (8) Thymus. (9) Mediastinal tumors.

For the examination of the abdominal organs, the precoccygeal or retropubic air injection is used. The stomach is distended with a carbonated drink. Films are exposed with the patient standing. The examination is useful in demonstrating the kidneys, suprarenals, the renal pedicle and pyelo-ureteric junction. Deformation of the kidneys and perirenal infiltrations and adhesions are well visualized. The splenic contours and perisplenic adhesions become visible. The external gastric contour, thickness of the gastric wall, adhesions, enlarged lymph nodes and pressure deformities are demonstrated. Morphologic changes of the pancreas and liver become clearly visible. Enlargement of lymph nodes becomes visible and indications for surgical treatment of tumors can be drawn more clearly. In cases of lesions in the neighborhood of the diaphragm, valuable information can be obtained (herniations, hydatid cysts, paravertebral abscess, neurinoma, pheochromocytoma, enlarged lymph nodes).

The method described is apparently used mainly in France and Italy, and according to the authors, represents a valuable contribution to the roentgenological examination of the mediastinal and abdominal structures.—*Karol E. Matzinger, M.D.*

STECKEN, A. Über Varizen der Lunge. (On varices of the lung.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Jan., 1955, *82*, 54–63.

The author reports 2 cases of varicosities of the lung in a fifty-one and a twenty-eight year old patient and reviews the literature.

In contrast to arteriovenous fistulae, there were no pulsations visible on fluoroscopy or kymography and nothing was heard on auscultation. In both cases, the right upper lobe was involved. Diagnosis was made by laminagraphy in the anteroposterior and lateral views, showing connection with the hilar vessels. In both cases, the findings were known but not diagnosed for over ten years. There were no clinical pulmonary findings in either case. Etiologically, a congenital vascular anomaly was assumed. In one case this was substantiated by observation of frequent familial vascular anomalies with nose bleeds and pulmonary bleeding.

The article contains seven excellent reproductions of chest roentgenograms and laminagrams.—*E. G. Wissing, M.D.*

GELLI, G. Micosi (candidosi) polmonare in forma cavitaria. (Particolare riferimento al trattamento chirugico.) (Pulmonary mycosis [moniliasis] in cavitary form; with particular reference to the surgical treatment.) *Riv. clin. pediat.*, Sept., 1954, *54*, 153–187.

A case of pulmonary mycosis with cavitation is reported in a girl ten years of age.

The mycete responsible for the disease was *Candida albicans*, species *C. stellatoidea*.

The diagnosis was based on the finding of mycetes in the sputum, serologic and allergic tests, studies of the action of the mycetes on experimental animals, the histologic report and the culture prepared from the gross specimen.

All of the usual therapeutic modalities (antibiotics, vaccines, novex, and potassium iodide) were ineffective and, in a relatively short time, lobectomy was performed with subsequent complete recovery.

The author points out the indications for the surgical treatment of the cavitary form of pulmonary mycosis as well as the other forms of the disease.

The surgical treatment referred to in this report was done for the first time in a child.—*Geno J. DiBagno, M.D.*

DEBORD, ROBERT A., and SIBILSKY, CARL E. Congenital obstructive emphysema treated by lobectomy. *A.M.A. Am. J. Dis. Child.*, Dec., 1954, *88*, 775–778.

A case of congenital obstructive emphysema in an infant, a two month old white boy, is described in this article. The authors report the case because of its interest to both the pediatrician and the surgeon. The case demonstrates how well infants tolerate major thoracic surgery.

The symptoms of obstructive emphysema usually occur early in life, sometimes within the first week after birth. Dyspnea, cyanosis and choking spells during feedings are the prominent symptoms. There may be respiratory wheezing. Physical examination reveals hyperresonance to percussion over the involved lung. The heart is pushed far over into the opposite pleural cavity. In the case reported in this article, the obstructive emphysema involved the left upper lobe.

The authors state that the roentgenogram of the chest is usually diagnostic. There are marked translucent areas in the lung which is involved. However, faint linear lung markings can be seen extending out from the hilus. The diaphragm is flattened and depressed. A marked shift of the mediastinum away from the involved lobe, and areas of atelectasis in the remaining good lung are seen.

Bronchoscopy will exclude intrabronchial foreign body or mucus plug in a bronchus, and a swallowing function examination with barium medium will differentiate the condition from diaphragmatic hernia. True bronchogenic cysts are usually solitary, are round and have a thicker wall, and may have a fluid level. The roentgenogram should be of assist-

ance in differentiating emphysema from spontaneous tension pneumothorax.

The authors list causes of the condition which have been discussed in previous articles in the literature: (1) deficiency or absence of the cartilaginous rings of the bronchus, resulting in abnormal flaccidity and collapse of the bronchus on expiration; (2) compression by an abnormal vascular structure, such as a persistent patent ductus arteriosus or abnormally placed vein; (3) redundant flap of bronchial mucosa, acting as a check valve; (4) congenital malformation in the intrauterine development of the lung buds. The last named factor is thought to be the most likely cause by the authors.

Lobectomy in the case reported was performed successfully and the patient made a good recovery with no further respiratory difficulty.—*Ralph S. Bromer, M.D.*

KEATS, THEODORE E., and CRANE, JACKSON F. Cystic changes of the lungs in histiocytosis. *A.M.A. Am. J. Dis. Child.*, Dec., 1954, *88*, 764–771.

The authors' review of the literature revealed 12 previously reported cases of histiocytosis with cystic, so-called "honeycomb" lungs. Of these, the Letterer-Siwe variety comprised the smallest group.

The case reported in this article is that of a white girl, of two and a half years, with primary pulmonary histiocytosis of the Letterer-Siwe form. The roentgenogram of the chest revealed generalized emphysema, with many areas of relative radiolucency interspersed with areas of linear infiltration, suggesting cysts or localized obstructive emphysema. Pathologically, the disease process was of unusual distribution, involving primarily the lungs and only focally infiltrating the thymus, thyroid, bone marrow and liver.

The authors suggest that multiple lung cysts evolve from necrosis of the nodular histiocytic infiltrates. They point out the necessity of considering histiocytosis as one of the possible, though rare, etiologies of cystic lung disease. As the multiple pulmonary cysts expand with entrapped air, these act as space-consuming lesions with respect to respiratory function, thus accounting for the marked dyspnea which these cases show.—*Ralph S. Bromer, M.D.*

LUCHERINI, T. Manifestazioni articolari e ginecomastia nell'osteoartropatia ipertro-fiz zante pneumica; studio del problema etiopatogenetico. (Articular and gynecomastic manifestations in hypertrophic pulmonary osteoarthropathy; study of the problem of pathologic etiology.) *Recenti progr. med.*, Sept., 1954, *17*, 256–280.

The author discusses at some length the etiologic factors and the clinical picture of hypertrophic pulmonary osteoarthropathy with particular reference to the articular manifestations and the occurrence of gynecomastia in some cases.

Two cases of hypertrophic pulmonary osteoarthropathy are presented, one without and one with gynecomastia. A biopsy of the mammary gland was done in the latter case and showed evidence of hormonal dysplasia while a biopsy of the synovial membrane of the knee in the former case showed inflammatory changes.

The author theorizes that the pathogenesis of hypertrophic pulmonary esteoarthropathy is related to endocrine factors at work within the anterior pituitary. He further theorizes that there is an increased production of S.T.H. (somatropic hormone) which is sometimes associated with the presence of gynecomastia.—*Geno J. DiBagno, M.D.*

BOUCOT, KATHARINE R., CARNAHAN, WILLIAM, COOPER, DAVID A., NEALON, THOMAS, JR., OTTENBERG, DONALD J., and THEODOS, PETER A. Philadelphia pulmonary neoplasm research project; preliminary report. *J.A.M.A.*, Jan. 29, 1955, *157*, 440–444.

In order to increase the curability of cancer of the lung, the characteristics of "curable" bronchogenic carcinoma must be delineated. In order to do this, the Philadelphia Pulmonary Neoplasm Research Project was set up in December, 1951. This unit plans to study 6,000 men over forty-five for a period of ten years by taking 70 mm. photofluorograms every six months and by studying their symptomatology and smoking habits. The patients are obtained from various sources; some are referred by private physicians, some by industry, some by clinics and some appear on their own initiative.

This is a preliminary report based on analysis of the first 3,945 cases. On the basis of either the first films or the presence of symptoms at the time of the first interview, 37 proved primary lung cancers were found. This is a prevalence of 0.9 per cent (0.3 per cent in those under 55 and 1.5 per cent in those over 55).

A reading of "suspect neoplasm" was made in 2.2 per cent of the cases on the initial photofluorogram. In this group were found 26 of the proved lung cancers. Of the remaining 11 cases, 9 were interpreted as exhibiting other diseases and 2 were erroneously read as normal. (The latter were picked up by the routine semiannual checkup in one instance and because of symptoms in the other).

Of the total group 85.8 per cent were smokers and suffered 100 per cent of the proved lung cancers. The incidence of carcinoma rises with the increase in the amount of smoking from 0.4 per cent for occasional to 1.3 per cent for heavy smokers.—*Myer Sharpe, M.D.*

Lemmon, Mark L., Kirklin, John W., and Dockerty, Malcolm B. Intralobar bronchopulmonary sequestration studied by multicolored vinyl acetate cast: report of case. *Proc. Staff Meet., Mayo Clin.*, Dec. 8, 1954, *29*, 631–637.

This anomalous defect consists of a large elastic artery that arises from the lower part of the thoracic aorta, the upper part of the abdominal aorta or the celiac axis. The artery originates most frequently from the left side of the aorta and enters the lower lobe of the lung through the inferior pulmonary ligament. Embryologically the vessel is the result of the persistence of one or more connections between the dorsal aorta and the postbranchial pulmonary plexus of the embryo. The earlier it arises during embryonic growth the greater the amount of pulmonary tissue it supplies. The pulmonary defect is intralobar and consequently both normally aerated pulmonary tissue and non-functioning, usually unaerated pulmonary tissue exist within the pleura of the involved lobe. The sequestered portion contains a bronchogenic cyst or cysts, which present subpleurally as soft rubbery nodules, or a system of bronchi which usually does not communicate with the normal bronchial tree. Unless infection occurs, the cysts are filled with a thick, brown mucoid material.

The authors' case presented multiple rounded densities in the right lower lobe. Following right lower lobectomy, vinyl acetate injection of the anomalous artery and the pulmonary artery and vein was performed. No anastomosis between the anomalous artery and pulmonary artery was demonstrated. The anomalous artery was confined to the sequestered segment of the right lower lobe and venous drainage was through the pulmonary vein.—*Arthur B. Smith, M.D.*

Lalli, Anthony, Carlson, Ralph F., and Adams, W. E. Intralobar pulmonary sequestration; report of three cases, with two additional cases of agenesis of the right upper and middle lobes in combination with anomalous systemic vessels. *A.M.A. Arch. Surg.*, Dec., 1954, *69*, 797–805.

Intralobar pulmonary sequestration, as described by Pryce, consists of an anomalous vessel running from the aorta in the vicinity of the diaphragm and a congenital bronchopulmonary sequestration or ectopia in the area of the lung served by the anomalous vessel. He felt the abnormal artery might supply only normal lung tissue, sequestrated lung alone, or a combination of both. The sequestration consists of bronchogenic cystic disease, usually having no communication with the normal bronchi. Extralobar sequestration or lower accessory lung differs in being a separate portion of lung, with a hilus attached to the parietes, esophagus or diaphragm, a separate bronchial system and venous drainage usually to the gastrointestinal or azygos systems. The etiology of intralobar sequestration lies in the persistence and traction effect of a lower accessory pulmonary artery arising from the primitive splanchnic plexus and "capturing" an end-bud of normally developing pulmonary tissue.

The authors present 3 typical cases of this disorder, with 2 additional cases with anomalous vessels from the aorta in combination with agenesis of the right upper and middle lobes. The latter 2 cases showed mediastinal shift and bronchography demonstrated the agenesis, whereas in intralobar sequestration, direct evidence is seldom gained by bronchography.—*Arthur B. Smith, M.D.*

Ciarpaglini, L., and Fusi, G. Considerazioni sulle possibilità e sui vantaggi della stratigrafia in confronto alla broncografia nella diagnosi dei tumori del polmone. (Considerations on the possibilities and the advantages of laminagraphy compared to bronchography in the diagnosis of lung tumors.) *Ann. ital. di chir.*, 1954, *31*, 589–639.

The authors, after a brief review of primary carcinoma of the lung from an anatomic and roentgenologic point of view, discuss in some detail the actual possibilities of laminagraphy as contrasted to bronchography in the roentgen diagnosis of this affection.

A variety of cases (total of 7) were seen by the authors at the Radiologic Institute of the University of Firenze, most of which had laminagraphic and bronchographic studies directly indicated by symptomatology; some, however, were picked up on routine survey with no symptoms referable to the chest.

Bronchoscopy was done on all of the cases and most of them were subsequently explored surgically.

Bronchography and laminagraphy gave comparable results in those cases affecting the main stem bronchi and where there was involvement of a lobar type, while, in those lesions involving the bronchopulmonary segments bronchography was of more value. On the other hand, laminagraphy was particularly useful in the study of parenchymal opacities and in the recognition of hilar and mediastinal tumefactions.

The authors conclude that the two methods of study are complementary and, in general, should be used together.—*Geno J. DiBagno, M.D.*

Siegal, William, Plunkett, Robert E., and Locke, Ben Z. Case findings from routine chest roentgenograms; mass surveys of communities versus general hospital admissions. *J.A.M.A.*, Jan. 29, 1955, *157*, 435–440.

Since 1947, 2,146,231 persons in upper New York over 15 years of age have had screening chest roent-

genograms (either 4 by 5 inch or 70 mm. films) under the auspices of the Bureau of Case Finding of the Division of Tuberculosis Control of the New York State Department of Health. Of these, 1,169,715 have been examined in mass surveys by mobile equipment and 1,076,506 in 71 general hospitals by fixed equipment. The latter include both in- and outpatients.

The sexes were equally divided in the mass surveys while in the hospital programs two-thirds were females. The average age was thirty-seven for females in both programs and thirty-eight and forty-six respectively for males in the mass survey and hospital programs.

On the initial examination (which included a 14 by 17 inch film in the suspicious hospital cases), the cases were categorized as probably active, probably inactive and suspected tuberculosis. The hospital rates were always higher than the mass survey rates; thus, in the hospital program, the total rate was 15.9 per 1,000 persons and in mass surveys 9.3 per 1,000 persons with corresponding rates for probably active tuberculosis of 2.7 for hospitals and 1.7 for mass survey.

Analysis indicated that 83 per cent of the hospital and 86 per cent of the mass survey cases had never been previously reported. Of these groups, 7,996 hospital cases (69 per cent) and 5,620 mass survey cases (71 per cent) were followed up clinically.

Active tuberculosis was found in 1,568 of the hospital group, a rate of 1.7 new cases for every 1,000 persons examined roentgenographically. The corresponding figures for the mass survey group were 1,254 and 1.3 per every 1,000 persons examined roentgenographically. Thirty-one per cent of those found through the hospital programs were hospitalized for treatment as compared with only 12 per cent in the mass survey study. This reflected the fact that 2½ times as many hospital as mass survey cases were initially classified as far advanced.

The initial roentgen estimation of the state of activity of the disease was found to be correct in 55.1 to 63.8 per cent of the cases.

In 1953 the screening roentgenograms were analyzed for suspected intrathoracic tumors among other conditions. A rate of 3.7 per 1,000 persons was found. This is 1½ times the rate for suspected active tuberculosis. The rate for males was 5.8 and for females 2.3, a ratio of 2½ to 1. Follow-up is incomplete but spot checks seem to show that 10 per cent of the suspects were found to have bronchogenic carcinoma. —*Myer Sharpe, M.D.*

KREMENS, VICTOR. Demonstration of the pericardial shadow on the routine chest roentgenogram: a new roentgen finding; preliminary report. *Radiology*, Jan, 1955, *64*, 72–80.

Visualization of the pericardial shadow along the left cardiac contour on the routine posteroanterior chest roentgenogram is possible in a small percentage of normal individuals. Clear and unequivocal demonstration is probably not obtained in more than 5 per cent, while less clearly defined or less complete demonstration may be encountered in an additional 5 per cent. Demonstration of this silhouette of the pericardium is made possible by the subepicardial fat layer which appears as a curvilinear shadow of increased radiolucency between the density of the myocardial mass and the adjacent parietal pericardium.

The normal pericardial shadow varies in width from a fraction to 1 or 2 mm. A pericardial shadow of more than 2 mm. in width is indicative of pericardial thickening, although a similar widened shadow lateral to the subepicardial fat layer may be seen in some instances of small or moderate pericardial effusion. When a thickened pericardial shadow is demonstrated, serial laminagraphic exposures are of value in providing additional evidence of the abnormality and in following the course of the disease and response to therapy.

A case of pericarditis in which the roentgen demonstration of a thickened pericardial shadow was of value in establishing the diagnosis and in following the course of the disease is presented.—*Harold C. Hamilton, M.D.*

HAIGHT, CAMERON, SLOAN, HERBERT, and RUBEL, WILLIAM F. Coarctation of the aorta. *J. Michigan M. Soc.*, Jan., 1955, *54*, 41–44; also, 56.

The authors review their experience with 30 cases of coarctation of the aorta operated upon between January 1, 1948 and October 1, 1953. Seventeen were males and 13 were females ranging in age from seven months to thirty-nine years with a mean age of 19.7 years. The operation in 28 cases was excision of the area of coarctation followed by end-to-end anastomosis; in 1 instance an aneurysm immediately distal to the coarctation was found and, at a subsequent operation, excision of both abnormalities followed by insertion of a graft was performed; in the other case, lack of collateral vessels made a definite procedure hazardous and the chest was closed.

The results in 20 cases (66.6 per cent) were judged excellent (symptoms and hypertension being relieved). In 7 others (23.3 per cent), symptoms were either completely, or almost completely relieved and the blood pressure did not exceed 155 systolic or 100 diastolic. These were considered good results. Of the remaining 3 cases, 1 had only a fair reduction in hypertension but complete relief of symptoms, another had no symptomatic relief but a delayed lowering of blood pressure, and the third was the patient for whom only the exploratory procedure was done.

The authors now believe that operation should be advised for most patients, especially those in the younger age groups (ideally in the second decade)

unless there is some recognized contraindication. Angiocardiography was used preoperatively in all cases to evaluate the anatomical situation and postoperatively in cases with unsatisfactory results.—*Myer Sharpe, M.D.*

Abdomen

Bulgrin, James G., and Holmes, Francis H. Eventration of the diaphragm with high renal ectopia; a case report. *Radiology*, Feb., 1955, *64*, 249–251.

The authors report a case of eventration of the diaphragm with an associated high renal ectopia. This condition is reported because it is very rare and more often overlooked than is generally realized. The embryology is reviewed, emphasizing the relationship of development of the two structures. Figures of two roentgenograms are included.—*E. E. Seedorf, M.D.*

Hillemand, P., and Barré, Y. A propos de l'étiologie de certaines hernies diaphragmatiques par l'hiatus oesophagien. (On the etiology of certain diaphragmatic hernias through the esophageal hiatus.) *Presse méd.*, Dec. 25, 1954, *62*, 1791–1793.

The authors were impressed by the frequency of spinal anomalies and endocrine disturbances in their patients (77 per cent of 70 patients).

The following spinal lesions were demonstrated roentgenologically: hypertrophic arthritis, kyphoscoliosis with and without spur formation, wedge-like deformity of the vertebral body and arthritis of the costotransverse articulations. These lesions, according to the authors, might cause herniation by increasing abdominal pressure as in kyphoscoliosis or by decreasing the tonus of the muscles of the esophageal hiatus. This latter might be caused by altered innervation of the diaphragm due to inflammation or compression of the nerve fibers secondary to changes of the spine.

Treatment in these cases attempts to eliminate spinal inflammatory changes and to increase the tonus of the hiatus musculature by ionization and electrotherapy.

Endocrine disturbances, concerning both the gonads and the thyroid, were also frequently observed in these patients. Hypothyroidism was found in both male and female patients, in the latter often associated with menopausal syndromes.

Of 21 patients treated with desiccated thyroid, 16 showed marked improvement. Of these 8 showed decrease or disappearance of the hernia on roentgenological examination and 3 disappearance of esophageal reflux with persistence of herniation.—*Karol E. Matzinger, M.D.*

O'Connor, Francis J., and Ritvo, Max. Diagnosis of hiatus hernia on plain roentgenograms of thorax and abdomen. *J.A.M.A.* Jan. 8, 1955, *157*, 113–117.

The authors emphasize the importance of identifying the structures visualized through the cardiac shadow. They observe one or two esophageal hiatus hernias each week at the Boston City Hospital from routine chest examinations.

The criteria for such a diagnosis are clearly outlined and their clinical significance discussed.—*V. L. Peterson, M.D.*

Whitehouse, Walter M. Correlation of surgical pathology with telepaque cholecystography in doses of two grams. *Surg., Gynec. & Obst.*, Feb., 1955, *100*, 211–215.

The author reports 124 cases with correlation of roentgen studies and surgical findings. A dose of 2 gm. of telepaque was used. Roentgen evidence of gallbladder abnormality was demonstrated in 123 cases. At operation, the other patient was shown to have slight chronic cholecystitis without calculus.

Roentgen evaluation of the gallbladder is based on: (1) presence or absence of gallbladder visualization; in the former case, the shadow is considered as of excellent, good or faint density; (2) when demonstrated, the type of calculus present.

There were 50 cases (40.3 per cent) with non-visualization of the gallbladder. Forty-five cases showed presence of calculi of which 6 were opaque and were demonstrated preoperatively. Two cases of non-visualization were due to carcinoma of the gallbladder and head of the pancreas, respectively. There were 27 cases with faint visualization, 44 cases with good visualization and 3 cases with excellent visualization of the gallbladder.

Histologic evidence of cholecystitis was found in all gallbladders which were not visualized with telepaque or whose shadows were faint. The striking finding was the occurrence of varying degrees of chronic cholecystits in 43 out of 44 gallbladders with good visualization, and in 3 cases showing excellent visualization. This indicates that considerable gallbladder function, evidenced by the concentration of the contrast medium, is retained despite the presence of chronic cholecystitis. However, the more severe inflammatory changes are reflected in the decreased intensity of the gallbladder shadow. The presence of calculi also reflects the underlying changes.

In cases of non-visualization re-examination with the same dose confirmed the original finding. Increasing the dose on re-examination is of no particular value in gallbladder visualization, although more calculi may be demonstrated.

When considering the decreased density of the gallbladder shadow as indicative of cholecystitis and probable presence of calculi, and the presence of cal-

culi in the better visualized gallbladder as indicative of inflammatory changes, a high degree of accuracy in the evaluation of gallbladder disease can be maintained with telepaque oral cholecystography. —*William H. Shehadi, M.D.*

GAYLIS, H., and GUNN, KENNETH. Transduodenal cholangiography. *Surg., Gynec. & Obst.*, Feb., 1955, *100*, 249–253.

Transduodenal cholangiography is described as a new procedure for the visualization of the biliary tract by retrograde filling through the duodenum. This was accomplished by the introduction of a special "double ballooned" tube (modified Miller-Abbott tube) under fluoroscopic control into the second part of the duodenum. When inflated by air this more or less obstructs the duodenum above and below the location of the papilla of Vater. The injection of iodized solution into the duodenum through an opening in the tube between the distended balloons resulted in retrograde filling of the biliary tract. This was successful in a patient who had prior surgery with anastomosis of the bile ducts into the duodenum.

Further evaluation of this technique was done on 5 normal subjects but with no success despite the use of antispasmodics. It was considered that the normal sphincteric function plus the "protective" effect of the folds of the duodenum precluded retrograde filling.

While this method may not be applicable in normal conditions, it should be useful in the investigation of cases in which the common bile duct has been anastomosed to the duodenum, where sphincterotomy has been performed or when spontaneous internal biliary fistulas have occurred opening into the duodenum or stomach.

Reproduction of 4 roentgenograms, 2 photographs of the special tube and a photomicrograph of a section of the termination of the common bile and pancreatic ducts into the duodenum accompany the article.—*William H. Shehadi, M.D.*

TESCHENDORF, W. Visualization of the biliary tract by means of biligrafin, especially after cholecystectomy. *Am. J. Digest. Dis.*, Sept., 1954, *21*, 247–251.

"Biligrafin" (Schering) now extensively used for the visualization of the biliary tract has been available only in 20 per cent concentration. A new preparation of "biligrafin," a 40 per cent compound of an iodolithium salt, known as "biligrafin forte," is now being tested for clinical use. Teschendorf has employed the new medium in 200 patients, mostly in individuals where the gallbladder has been removed or is no-function by routine cholecystography.

The dose of 20 cc. of "biligrafin forte" is injected slowly (8 minutes for injection with a pause of 1 to 2 minutes halfway through the injection period). In only 1 of the 200 cases was any reaction recorded, and this was mild in nature.

The cholangiographic film studies are made as with "biligrafin," *i.e.*, 10, 20, 30, and 40 minutes after injection.

This medium allows visualization of the hepatic and common bile ducts in 98 per cent of cases, regardless of the presence or functional status of the gallbladder.

Typical cases of biliary tract disease studied by "biligrafin" and "biligrafin forte" are illustrated and the indications for use of these media discussed. —*Harold Fulton, M.D.*

ANACKER, H. Die Füllungs- und Entleerungsvorgänge in den Gallenwegen. (The filling and emptying functions in the bile ducts.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Aug., 1954, *81*, 143–150.

It is now possible to observe the filling and emptying of the gallbladder as well as of the bile ducts under physiological conditions with the use of biligrafin. The author reports the following observations:

The bile, which is continuously excreted by the liver, reaches the main hepatic duct and from there it may fill the gallbladder and the common duct at the same time, or the gallbladder filling may be delayed for some time after the common duct is well visualized. At times the gallbladder image is only demonstrated after some of the contrast medium has passed into the duodenum.

The filling of the gallbladder occurs in two different ways. The contrast bile is either distributed homogeneously through the gallbladder from the start and the density increases steadily, or the filling occurs segmentally from the infundibulum to the fundus. Occasionally, after the gallbladder is completely visualized, stratification of the contrast medium occurs in the erect posture, simulating floating cholesterin calculi. This stratification disappears when the bile is well mixed with the medium after several bending exercises.

There is no demonstrable evidence that the bile ducts react in any way to the stimulus of a fatty meal. On the other hand, they do empty under the influence of internal pressure and gravity of the bile, especially in the erect posture. Under these circumstances, a contraction has been observed in the papillary end of the common duct. In 1 exceptional case out of 80 examinations active contraction has been noted in the small bile ducts during an attack of pain. The patient, who had a choledochoduodenostomy because of gallstones, suffered occasional short lasting attacks of sudden pain in the right upper quadrant. The emptying of the gallbladder, especially its response to fat stimuli, is well known. In this connection a reflux of the bile into the hepatic duct was observed whenever the pressure in the

common duct was greater than in the hepatic duct.

The question whether the Bernhard's restraint mechanism, a circular spasm in the hepatic duct to prevent the reflux of bile from causing liver cell damage, occurs every time the gallbladder contracts has not been determined during these observations.—*S. Silvera, M.D.*

Lange, L. Simultane Cholecysto-Cholangiographie (Vorläufige Mitteilung). (Simultaneous cholecysto-cholangiography; preliminary report.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Aug., 1954, *81*, 150–155.

A technique is described by which both the gallbladder and the biliary ducts are demonstrated on the same film.

It is known from recent publications that the biliary ducts visualize first and the gallbladder 15 to 150 minutes later, after the intravenous injection of 20 to 40 cc. of biligrafin. By using the optimum time the author was able to devise a cholecystocholangiography. He makes two 20 cc. injections so as to visualize the gallbladder with the first injection, ignoring the cholangiography that occurs during this procedure, and visualizes the biliary ducts with the second injection after the gallbladder is well demonstrated. This is only possible providing that the liver is able to excrete the contrast medium, that the cystic duct is patent and that no complete obstruction of the extrahepatic bile ducts is present.

The technique used by the author is as follows: The patient is prepared with cleansing enemas the evening before the examination. Twenty cc. of biligrafin is injected intravenously. The patient is kept on the right side. The second injection of 20 cc. biligrafin is made 2½ to 3 hours after the first injection. Exposures are made in the prone position with the right side of the body elevated about 45 degrees with the following factors: 40 inches target skin distance, 55 to 70 kvp., 250–400 milliampere seconds. In case the gallbladder or the biliary ducts are superimposed with gas or fecal contents, laminagraphic studies can be made. The author encountered no ill effects from this procedure. However, in the event of cardiovascular collapse, as reported in 2 cases by Hagedorn ¾ to 1 hour after the injection of 20 cc. of biligrafin, the examination is interrupted and no additional biligrafin is injected.—*S. Silvera, M.D.*

Rosenbaum, Franz-Josef. Die laparoskopische Cholangiographie. (The laparoscopic cholangiography.) *Klin. Wchnschr.*, Jan. 1, 1955, *33*, 39–41.

The laparoscopic cholangiography gives better diagnostic information than other methods. Technique of the laparoscopic cholangiography is described as follows:

The gallbladder is punctured with the needle, not from the free peritoneal cavity, but always through the liver and gallbladder bed with a canula 25 cm. in length and 2 mm. in caliber. Of the contents of the gallbladder, as much as possible, in the author's cases 5–250 cc. is aspirated, and the gallbladder is filled with 20 to 40 cc. of per-abrodil. After one or two scout films, further films before and after the fatty meal are exposed. The puncture of the gallbladder through the liver and gallbladder bed is preferred because the liver rapidly covers the puncture opening and prevents peritonitis from leaking of the contents of an infected gallbladder into the peritoneal cavity.

Six cases are described and illustrated: Cholelithiasis with stone occluding the cystic duct; cystic duct occlusion by a cherry-size stone; obstruction of the choledochus by carcinoma of the duct; occlusion of the papilla vateri by carcinoma of the head of the pancreas; incomplete obstruction of the choledochus by pressure of an extrinsic lesion and by a stone close to the papilla. In one case a laparotomy nine days after laparoscopic cholangiography revealed a subphrenic abscess. In this case the puncture had been performed through the liver, but not through the gallbladder bed, which prevented an immediate covering of the puncture by the liver.

The author is very satisfied with results of the laparoscopic cholangiography by which in many cases a laparotomy can be avoided.—*Hans Lewin, M.D.*

Twiss, J. Russell, Gillette, Lee, Beranbaum, Samuel L., Poppel, Maxwell H., and Hanssen, Eilif C. Postcholecystectomy oral cholangiography. *A.M.A. Arch. Int. Med.*, Jan., 1955, *95*, 59–65.

The author reviewed 106 oral cholangiograms performed by the following technique: Dehydration was obtained by withholding all liquids for twelve hours preceding the examination except for a glass of hot water with each dose of the opaque medium. A double dose was used in all cases. This was given either the evening before or the morning the films were taken. Camphorated opium tincture was given two hours after the opaque medium. After visualization of the bile ducts, an inhalation of amyl nitrite and/or the ingestion of a fat meal was ordered and a film was taken thirty minutes later.

Visualization of the bile ducts has been obtained in 72 per cent of the cases in this series. Pathologic conditions such as stones and cystic duct remnants have been visualized and proved by operative findings.—*James J. McCort, M.D.*

Davis, Lawrence A., Knoefel, Peter K., and Pirkey, Everett L. Factors influencing the roentgen visualization of the gastric mucosa, *Radiology*, Jan., 1955, *64*, 29–33.

This study was undertaken in an attempt to de-

termine (a) whether various patient factors, such as age, weight, sex, and the composition and volume of the resting gastric juice, had any effect upon the visualization of the rugal pattern of the stomach by the opaque medium, and (b) whether particle size of $BaSO_4$ greater than that required by the U.S.P. influenced the pattern.

Barium sulfate preparations with a particle size greater than that of U.S.P. $BaSO_4$ were found to be undesirable. The greater the volume of resting gastric secretion, and the higher the total mucin content, the poorer was the rugal pattern obtained. They found no apparent correlation of age, weight, height or gastric acidity, with the class of rugal pattern observed.—*A. W. Sommer, M.D.*

FRIEDLANDER, F. C., and KUSHLICK, P. Trichobezoar. *Arch. Dis. Childhood*, Dec., 1954, *29*, 556–560.

Until recently only 400 cases of this rare condition had been described in the literature. The authors report the first case so diagnosed at the Transvaal Memorial Hospital for Children.

Bezoars (old Persian term meaning antidote to a poison) fall essentially into three groups: (A) trichobezoars, made of hair; (B) phytobezoars made of vegetable matter; (C) concretions.

The commonest are the trichobezoars; these occur in the younger age group, mostly the first and second decades, and predominantly in females.

From the point of view of etiology and treatment they present a two-fold problem: (1) The mechanical, the retention of hair in the stomach and the formation of a mass, usually a cast of the stomach requiring surgical removal, and (2) the motivating psychological factor behind the ingestion of hair—related to nail biting and tongue sucking, requiring psychiatric therapy.

While made mostly of hair trichobezoars may have intermeshed fibers from blankets and other fabrics. Phytobezoars are composed of vegetable matter mostly persimmons. They are commonest in males occurring in the fourth, fifth and sixth decades and are rare in childhood.

Concretions are formed after ingestion of furniture polish by furniture workers, usually consumed for its alcoholic content. Salol and other medicaments also contribute to the formation of concretions.

The main symptoms are pain, usually colicky, also anorexia, nausea, vomiting and at times diarrhea. Despite occasional occurrence of weight loss, general malaise and weakness, the patient's general condition is surprisingly good. A palpable epigastric or upper abdominal mass is the only striking finding. The diagnosis is made roentgenologically. Trichobezoars may cast a shadow on a plain film. Orally administered barium flows over and around the mass, presenting a reticular pattern, forming a cast of the stomach. Barium soaks into the mass while the stomach wall appears normal. Phytobezoars are smaller, produce a smooth filling defect and do not retain the barium.

The authors report a case of a nine year old white girl who for some time suffered attacks of colicky abdominal pain, nausea but no vomiting. The mother had noticed a palpable abdominal mass, confirmed by physical examination. From infancy, the patient picked and swallowed the fluff off her blankets. More recently, she had been sucking, biting and swallowing the long strands of her hair. She was a nail biter, but had no other psychological abnormalities. At school, she was one year ahead of her age group.

Physical examination revealed a painless firm sausage-shaped upper abdominal mass, the upper border disappearing under the rib margin. Roentgenographic studies demonstrated the presence of a mass occupying the entire stomach. Barium outlined the mass and left the stomach without delay. A diagnosis of bezoar was made. Laparotomy was performed and the mass was removed through a 3 cm. anterior longitudinal incision in the stomach wall. The gastric mucosa was hypertrophied, the rugae well formed. The postoperative course was uneventful. Reproductions of 3 roentgenograms and of 1 photograph of the removed specimen accompany the article.—*William H. Shehadi, M.D.*

OTTOMAN, RICHARD, E., and WOODRUFF, JOHN, H., JR. Polypoid diseases of the stomach. *Radiology*, Jan., 1955, *64*, 34–44.

The phrase "polypoid disease" is purely descriptive, indicating a variety of gastric diseases producing polypoid defects in and beneath the gastric mucosa. The wide variety of gastric polypoid lesions range from a sessile configuration through complete pedunculation. Conversion from a wholly sessile plaque to a pedunculated configuration may be brought about by the muscular movement of the stomach, aided perhaps by gravity.

Histologically these lesions may be segregated into several categories:

1. Inflammatory
   a) Specific
   b) Nonspecific
2. Tumors
   a) Benign
   b) Malignant
3. Miscellaneous

The neoplasms represent the largest and most important group. Serious complications are uncommon in the inflammatory group and the miscellaneous group comprises rare, non-neoplastic lesions which are important only in differential diagnosis.

Symptoms produced by polypoid diseases are vague, or even non-existent, until complications occur. Obstruction or bleeding may direct attention to such lesions early. Since there is a high incidence of polypoid lesions in the antrum and prepyloric

area, these tumors are prone to prolapse into the pylorus, with subsequent intermittent partial or complete obstruction. Later, ulceration may occur, with massive bleeding similar to that seen in a peptic ulcer.

The incidence of these lesions varies according to the examiner, type of examination, and patients selected for study. In autopsy material there is a greater incidence of benign polypoid neoplasms. This is indicative to some degree of the incidence of nonsymptomatic lesions. The importance of discovery and identification of polypoid gastric lesions lies in the tendency for the benign gastric neoplasms to undergo malignant change, or, in an association of benign and malignant lesions.

Several points of differentiation between the various polypoid gastric neoplasms are presented. However, specific identification of these lesions is extremely difficult since they so closely resemble each other. In general, a static or slowly growing lesion is benign. Its borders are regular, smooth, and occasionally nodular, rounded and sharply circumscribed. A stalk is frequently present. The rugae are generally normal but may be thin over the tumor surface. Margins are more apt to be irregular and less well defined. Pedicle formation is infrequent. Ulcers may occur. Most significant is the absence or reduction of peristalsis and loss of pliability of the gastric wall at the site of involvement in malignant neoplasms.—*Michael J. Healy, M.D.*

Ludin, H. Kombinierte Pankreaspneumographie. (Combined pneumography of the pancreas.) *Schweiz. med. Wchnschr.*, Jan. 15, 1955, *85*, 55–61.

A combination of retroperitoneal and intraperitoneal pneumography with sagittal laminagraphy is used for visualization of the pancreas. So far 8 cases were examined in this manner and 3 cases are presented; 1 case of carcinoma of the head of the pancreas, the other, a cyst of the tail of the pancreas and 1, a normal pancreas.

Oxygen is injected into the retroperitoneal space in the knee-elbow position under fluoroscopic control. This is followed by intraperitoneal injection of CO_2 in order to fill the omental bursa with gas and outline the anterior border of the pancreas. Laminagraphy is then performed with the patient sitting and bent forward with his right side against the film. A survey film is taken in the supine position. In 2 cases the omental bursa failed to fill with gas due to closure of the foramen of Winslow by adhesions.

As far as can be determined from such a small series of cases, the procedure is without danger and offers an excellent means of demonstrating pancreatitis, tumors and cysts of the pancreas.

The roentgen anatomy of the upper abdomen is discussed in detail and reproductions and tracings of the roentgenograms in 3 cases are presented.—*Frederick M. Reis, M.D.*

Wilson, John W. The diagnosis of abdominal cysts in infants and children. *Radiology*, Feb. 1955, *64*, 178–190.

The determination of the exact type of an abdominal cystic mass in a child is extremely important to the physician, to enable him to elect the proper course of treatment. The roentgenographic features can be a valuable adjunct to the clinician's findings, and the author discusses and correlates these various diagnostic features.

Roentgenographically a cyst is sharply outlined by smooth margins and tends to be symmetrical and round. This outline may change with position, and exposures in various positions can be helpful in differentiating a cyst from solid masses, which do not thus change in contour.

The essential clinical findings and the details of roentgenographic differentiation and diagnosis for each type of abdominal cyst are given. Their location, alterations in adjacent structures, communications, and specific roentgen appearance are detailed, and the methods for demonstrating these features presented (such as direct and indirect opacification, gastrointestinal series, abdominal films, cystograms, etc.).

The following cystic abdominal masses can occur:

1. Omphalomesenteric duct cysts, arising from a failure of complete obliteration of the embryonic vitelline duct.

2. Enterogenous cysts, arising anywhere along the gastrointestinal tract by duplication of a segment.

3. Mesenteric and omental cysts, both arising from lymphatic channels and differing only in their site of origin.

4. Pancreatic cysts, which can be true cysts (resulting from blockage of a major duct) or pseudocysts (probably subsequent to abdominal trauma). Rarely echinococcal cysts are primary here.

5. Choledochal cysts, arising from a cystic dilatation of the common bile duct in a child.

6. Urachal cysts, developing from an incompletely obliterated urachus.

7. Ovarian cysts, which are unusual in children, especially under ten years of age. Dermoids make up 35 per cent of those that do occur.

8. Hydrometrocolpos, which is a cystic dilatation of the vagina and uterus in infants, and is caused by an imperforate vagina with excessive uterine secretion. Hematocolpos does not manifest itself until puberty.

9. The hydronephrotic kidney, which is the most common cyst in the abdomen.

10. Simple renal cysts and polycystic kidneys.

11. Cystic dilatation of segments of the gastrointestinal tract in intestinal obstruction (especially closed loop obstruction).

12. Hepatic cysts, including echinococcal cysts.

13. Unattached retroperitoneal embryoma, which is the third most common retroperitoneal tumor in childhood.

14. Meningoceles, which protrude into the abdomen.—*C. L. Stevenson, M.D.*

ROOT, GROSVENOR T., CHRISTENSEN, BURT H., and PETERSON, CARRELL A. Intussusception: an eight year survey. *Surg., Gynec. & Obst.*, Feb., 1955, *100*, 171–174.

The authors, from the Children's Hospital of East Bay, Oakland, California, report upon 66 children who were operated upon for intussusception. Most of the children were males under two years of age. Six of them developed intussusception in association with acute gastroenteritis. Four of the patients had undergone previous surgery for the same condition. The signs and symptoms were fairly classical. Three-fourths of the patients developed vomiting; two-thirds of them had a palpable mass and passed blood by rectum. Eighty per cent of the patients had leukocytosis.

Forty-four per cent of the patients had symptoms for less than twenty-four hours but 47 per cent had symptoms for more than forty-eight hours, and in the latter group one-fourth of them required bowel resection at the time of surgery. The authors emphasize that the farther the intussusception had progressed into the left side of the colon, the more likely was there necessity for bowel resection and the higher was the mortality rate.

The importance of barium enemas in diagnosis is stressed. Seventy per cent of the patients had roentgen diagnostic studies. In 3 instances the barium enema reduced the intussusception completely and in 9 instances the barium partially reduced the intussusception. No specific effort was made to reduce the intussusception at fluoroscopy. The authors doubt the wisdom of such reduction. Of those patients having intussusception into the left side of the colon, 10 were ileosigmoid, 2 were ileorectal and 3 were "double." Twenty per cent of these patients required bowel resection. There were 3 deaths in the entire series. It is of interest to note that at the time of operation, 6 patients had a Meckel's diverticulum. There was Meckel's diverticulitis in 4 cases. In 18 instances (27 per cent) the appendix showed evidence of acute inflammation and was removed.

The article brings out very well the importance of the duration of symptoms, the value of early diagnosis and the effect of left sided intussusception on the surgical prognosis.—*C. L. Hinkel, M.D.*

STEINBACH, HOWARD L., ROSENBERG, ROBERT H., GROSSMAN, MOSES, and NELSON, THOMAS L. The potential hazard of enemas in patients with Hirschsprung's disease. *Radiology*, Jan., 1955, *64*, 45–50.

It appears to have been established that enemas can precipitate severe and often fatal reactions in patients with Hirschsprung's disease. One investigator believes this to be due to water intoxication and points out the similarity in symptomatology between these patients and those having the syndrome of water intoxication. In support of this, serial plasma electrolyte studies following tap water enemas in several patients with megacolon demonstrated a rapid diffusion of water into the circulating fluid, causing a precipitous fall in plasma sodium, proteins, and specific gravity. The electrolyte dilution occurred within fifteen minutes in one case. He concluded that the essential disturbance producing reactions in these patients was a state of low sodium shock.

A combination of factors adds to the rate of absorption of fluid from the megacolon: (1) a much larger absorptive surface than normal colons, (2) multiple superficial mucosal ulcerations, (3) high intraluminal pressure, which is a result of: (a) the partial obstruction to evacuation produced by the hypertrophied muscular wall in the "aganglionic segment," (b) the use of a high hydrostatic pressure in introducing the enema to overcome the interference of large amounts of fecal material usually present, and (c) the frequent use of prostigmine in these patients, which increases the muscular contractions of the colon.

Water intoxication alone does not explain the reactions seen in some patients. Some of the reactions are similar to prostigmine poisoning alone. The possibility that they are due to reflex inhibition of the heart and shock produced purely by mechanical distention of the colon must be considered. Since the disease is primarily a neurologic disturbance, it is possible that an altered or exaggerated reflex could be produced, causing cardiac arrest and shock.

Regardless of the mechanism producing these reactions, it is important that measures be taken to prevent them, and to be able to treat them once they occur. The patient's general condition should be very satisfactory before giving the enema. It is desirable to use an isotonic saline solution for the enema to reduce the possibility of water intoxication. By the same token, as much fluid as possible should be recovered following the enema. It is not necessary, or desirable, to fill the entire colon in order to diagnose Hirschsprung's disease. The diagnosis rests on the demonstration of a narrowed rectum and rectosigmoid associated with marked dilatation of the descending and sigmoid colon. The hydrostatic pressure of the fluid being administered should not be excessive. Raising the enema can 2, and not more than 3 feet above the level of the body is sufficient. If a reaction occurs in spite of all precautions, the patient should receive symptomatic treatment for shock.—*Michael J. Healy, M.D.*

GENITOURINARY SYSTEM

HOPE, JOHN, W., and CAMPOY, FRANCISCO. The use of carbonated beverages in pediatric excretory urography. *Radiology*, Jan., 1955, *64*, 66–71.

A method of employing carbonated beverages to improve pediatric excretory urography is described. The technique calls for dehydration for only one feeding in infants and in older children nothing by mouth for twelve hours before examination. A preliminary roentgenogram is made and the contrast medium is administered intravenously. The children are immediately given a bottle or glass of Pepsi Cola. It is emphasized that personal attention of the doctor is necessary to see that sufficient carbonated beverage is ingested and retained and that roentgenograms are made at the most appropriate moment.

The use of this method has greatly increased the diagnostic quality of this important examination in infants and children.—*E. E. Seedorf, M.D.*

Moore, C. A., and Dodson, C. C. Urinary tract calculi in children; renal and vesical calculi in an eight-month-old child. *A.M.A. Am. J. Dis. Child*, Dec., 1954, *88*, 743–748.

In this article the literature referable to urinary tract calculi in infants is reviewed and, as indicated by autopsy series, the condition is not uncommon in certain regions of the world where nutrition is poor. However, in the United States the incidence is quite low and very few cases of urinary tract calculi in children under five years of age have been reported.

Malnutrition (deficiency of protein and water-soluble vitamins) is undoubtedly an important factor in the etiology of the condition. In some metabolic disturbance such as cystinuria, oxaluria, and lithuria, there is an increase in urinary crystalloids which frequently leads to the formation of calculi. Infections, especially urea-splitting organisms, produce an alkaline urine which favors the disruption of the crystalloid-colloid equilibrium and subsequent stone formation. Urinary stasis is frequently the predisposing factor in the development of infection. Randall has described submucosal calcific plaques which form in the tips of the renal papillae. Subsequently, these may break off and become deposited in the calyces or renal pelvis, where they act as a nidus for calculus formation.

The patient reported in this article was a white girl, eight months of age. The presenting symptoms were primarily gastrointestinal as is frequently the case in urinary tract disease in children under five years of age. Because of the presence of persistent pyuria, a roentgenogram of the genitourinary tract was secured which showed densities in the region of the left kidney. Complete urological studies revealed calculi also in the bladder. The calculi in the bladder were small and were removed through the cystoscope sheath. Later a left nephrectomy was performed because of infection. Complete recovery followed.

The authors emphasize the value of urological evaluation in children with unexplained fever associated with gastrointestinal symptoms and with or without pyuria.—*Ralph S. Bromer, M.D.*

Baron, George J., and Koenemann, Richard H. Arteriovenous fistula of the renal vessels; a case report. *Radiology*, Jan., 1955, *64*, 85–87.

A case of arteriovenous fistula of the renal vessels, the fifth which has been described in the literature, is reported. Congenital aneurysms, tumor, and trauma are listed as causes of this condition, and it is stated that a thrill or bruit over the affected kidney is a prominent clinical finding. This condition may be a rare cause of hypertension.

Pyelography may be used to indicate the site of the lesion, though this examination does not yield pathognomonic findings.

One should be able to actually visualize the arteriovenous communication by the use of angiography, so that a specific preoperative diagnosis could be made.—*A. Ralph Watson, M.D.*

Nervous System

Breit, A., and Wiedenmann, O. Ergebnisse der Gasmyelographie. (Results of gas myelography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Dec., 1954, *81*, 761–767.

Positive or negative contrast media can be used for visualization of the spinal cord. Pantopaque injection and discography are not generally accepted in Germany. Water-soluble iodine preparations are only recommended for examination of the lumbar spinal canal. Gas myelography was first reported by Dandy in 1919 (and 1925), then superseded by lipiodol injection, but Lindgren later advocated the complete replacement of spinal fluid by air. The authors have used this method since 1948, in 1952 and 1953, together with simultaneous laminagraphy.

Technique. Suboccipital puncture, injection of oxygen or air, 80 to 120 cc., slightly more gas injected than fluid removed in order to open subarachnoidal spaces by increased pressure. The examination includes:

1. Lateral roentgenograms of the lumbar, dorsal and cervical spine;
2. Laminagrams of the same;
3. Laminagrams of the same in the anteroposterior view.

Of 685 gas myelographies done, 207 were considered normal and 478 pathological. Findings included almost 350 cases of degenerative changes of the spine; 53 cases of extramedullary tumor formation; 24 cases of intramedullary tumor (in this group additional positive contrast myelography was necessary in 54 per cent); 25 cases of arachnoiditis (necessitating 85 per cent of positive contrast examination); etc. Ruptured discs were frequently demonstrated by laminagraphy alone.

In the discussion the authors state that pathology can be demonstrated in about 90 per cent of cases,

but that thin iodine contrast media are superior for determining the type of pathology, particularly in cases of tumor or ruptured intervertebral discs.—*E. G. Wissing, M.D.*

BRIERRE, J. T., and COLCLOUGH, J. A. Total myelography; complete visualization of the spinal subarachnoid space. *Radiology*, Jan., 1955, *64*, 81–84.

Confirming and expanding on techniques suggested by previous investigators, the authors have developed a method of total myelography which they feel is not only easier for the patient, surgeon, and radiologist, but also increases the accuracy of the examination.

Their technique consists of the injection of 21 cc. of pantopaque into the lumbar subarachnoid space in the lateral recumbent position, with the head slightly elevated. This is preceded by a routine spinal tap with pressure readings, observation of cerebrospinal fluid dynamics, and withdrawal of fluid for laboratory examination. After injection, the needle is withdrawn, the patient assumes the prone position with feet against a footboard, and the table is tilted vertically so that he may walk a few steps. Following his return, the lumbosacral region is examined fluoroscopically, and roentgenograms are made. After being returned to the horizontal prone position, he turns to the supine position with the head slightly elevated. This causes complete filling of the thoracic region so that it can be examined. He is then returned to the prone position, and with the neck in extension, the table is tilted head downward, not exceeding 45 degrees, and the cervical region is observed. The patient then sits up for a few minutes before another spinal tap when withdrawal of the medium is done in the lateral recumbent position.

The advantages of this method of examination are: (1) The ease of handling of the patient for lumbar visualization in the erect position. (2) The erect position and walking may cause lumbar disc protrusion when it might not otherwise be demonstrated. (3) By utilizing the supine position, the entire thoracic region is easily examined. (4) Excessive inversion of the patient in order to examine the thoracic and cervical regions is eliminated. (5) Removal of the radiopaque medium is much easier, when the large quantity is used. (6) There is less globulation of the medium when larger amounts are used.

With this method, it is felt that intervertebral disc herniation can be definitely shown or excluded. —*C. L. Stevenson, M.D.*

SKELETAL SYSTEM

MCCORMACK, L. J., DICKSON, J. A., and REICH, A. R. Actinomycosis of the humerus. *J. Bone & Joint Surg.*, Dec., 1954, *36-A*, 1255–1257.

Although the medical literature contains numerous references to actinomycosis, few cases have been reported which do not primarily involve the cervicofacial, thoracic, or abdominal regions of the body. Classification of cases of this disease has been based upon infection of these three areas. Primary involvement of the skeletal components of the extremities has occasionally occurred and this form has been the subject of two types of reports: cases of actinomycosis diagnosed by histological study and without culture, and in contrast, cases of proved actinomycosis of the knee joint, proved by culture. In the case reported in this article, the diagnosis was initially suspected on frozen section examination.

Roentgenographic examination of the right humerus of the patient, a male aged forty-four years, revealed the medullary cavity in the mid-shaft to be widened and irregularly radiolucent. Soft tissues about the region were swollen. In the right upper lung field, there was an increase in bronchial and vascular markings. There was a suggestion of a localized infiltrating lesion, involving the right upper lobe in its mid-portion, associated with hilar adenopathy. A biopsy was performed to clarify the diagnosis and as a result of the findings, the lesion was saucerized. Penicillin therapy was instituted, and a total dose of 34,800,000 units was given. He also received 60 drops of a saturated solution of potassium iodide in divided doses for almost four months. The patient has been asymptomatic for almost two years and roentgenograms made at the end of eighteen months showed an apparently healed lesion.

The Actinomyces have been divided into two species, the species *bovis* chiefly pathogenic for animals, and the species *israeli* chiefly pathogenic for man. In this case, the organism followed the growth pattern of *Actinomyces israeli* but the colonial morphology was that of *Actinomyces bovis*. The authors assigned a greater value to the appearance of organism in their case in solid media in classifying it as *Actinomyces bovis*.

Whether or not the lesion in the humerus was a primary actinomycosis was a difficult decision to make. The changes shown in the roentgenograms of the chest suggested the possibility of a primary involvement of the lung. However, the lesion in the chest was relatively minimal, with static findings for at least a year prior to development of the bone lesion. The patient could produce no sputum on numerous occasions, and cultures of gastric washings were negative. Clinically, the disease manifested itself as an osteomyelitis.—*Ralph S. Bromer, M.D.*

SMITH, A. MCEWEN. Sprain of the pisiform-triquetral joint. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 618–621.

Sprain of the pisiform-triquetral joint is a definite clinical entity. Six patients are reported in this article who were seen in a busy out-patient department during a period of two years. The pain was described as

aching in character, and was either brought on or made worse by exertion, especially heavy lifting. The insertion of the flexor carpi ulnaris was indicated as the site of greatest pain. All 6 patients were women between the ages of eighteen and thirty. In none did roentgen examination reveal any abnormality.

On clinical examination a constant finding was pain on resisted flexion of the wrist, greatest in the position of ulnar deviation. Pain was also elicited when the patients held a heavy object in the outstretched hand with the forearm in full supination. Passive movement of the pisiform bone on the triquetrum elicited pain of the same character as that complained of. The pisiform bone can normally be displaced laterally on the triquetrum in the position of ulnar deviation of the hand when the muscles are relaxed. Pain in this test is diagnostic of sprain of the pisiform-triquetral joint and in 2 of the cases movement was accompanied by crepitus.

The disability is such that in most cases operation is justifiable. Fusion of the pisiform-triquetral joint is preferred to excision of the pisiform because it restores stability to the wrist with the least disturbance to related structures.—*Ralph S. Bromer, M.D.*

Mason, Mark L. Traumatic dislocation of the hip in childhood. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 630–632.

Traumatic dislocation of the hip in a child is rare. The author found that the number of recorded cases in the literature in the last thirty-two years is 88. The youngest patient was aged eleven months and the oldest fourteen years. His case is a boy of one year and eleven months of age. One hour before being brought to the hospital by his father, the latter had tripped on the sand and had fallen on his son and partly buried the child in the sand. The dislocation was reduced under general anesthesia.

It is assumed by the author that the child was in the "knee-elbow" position on the sand, probably with his knees together, and the father landed heavily on his son's left buttock. The interest in this unusual injury lies in the development of avascular necrosis of the upper femoral epiphysis. Traumatic dislocation of the hip is necessarily accompanied by rupture of the ligamentum teres and tearing of the joint capsule. There is corresponding loss of blood supply in the region of the fovea and an impairment of the blood supply to the rest of the epiphysis. In the author's case, roentgenograms ten months after the injury showed no evidence of necrosis or other abnormality.—*Ralph S. Bromer, M.D.*

McCallum, R. I., Stanger, J. K., Walder, D. N., and Paton, W. D. M. Avascular necrosis of the femoral heads in a compressed air worker. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 606–611.

The authors state in their discussion of the case reported in this article that avascular necrosis of bone may occur from time to time without known cause, but the onset of severe destructive changes in the femoral heads of a man of thirty years of age within twenty-one months of work in compressed air suggests that this was the direct cause. Pain in the hips began only five months after his work in a tunnel had finished and working in the pressure he had "bends" affecting both lower limbs, though not to a degree that could be considered unusual in a compressed air worker. Roentgenograms of both hip joints showed gross destructive changes in the heads of both femora. The right acetabulum was normal and the only change in the left was a minor degree of lipping of the upper margin. Though the changes extended throughout the femoral head, the greatest deformity of the articular surface was on the superior aspect, which is normally subjected to the greatest weight-bearing pressure. The roentgenographic findings resembled those seen in avascular necrosis from severe damage to the blood supply to the femoral head and were not at that time characteristic of osteoarthritis. Roentgenograms of the rest of the skeleton showed no other lesions.

Decompression of the patient had been carried out according to regulation, but it is probable that bone lesions, like other forms of decompression sickness, cannot be prevented entirely by decompression according to standard tables, and that individual susceptibility plays a part. It is possible that bone disease occurs more frequently than is recognized and in cases of unexplained avascular necrosis, particularly in the humerus or femur, a history of work in compressed air should be inquired into.

Judet arthroplasty is recommended as of particular value in a case of the type reported because the femoral head has been destroyed. Such arthroplasty permits complete study of the damaged head, previously impossible. However, although arthroplasty is undoubtedly the operation of choice in bilateral disease, arthrodesis might still be the correct procedure in a unilateral case.—*Ralph S. Bromer, M.D.*

Lyford, John, III. The fate of femora with pathologic fractures due to malignant tumors, stabilized by intramedullary nails, as shown by serial roentgenograms, *Radiology*, Feb., 1955, *64*, 197–200.

Femoral stabilization with intramedullary nails is a valuable adjunct in the management of pathologic fractures of the femur.

Two cases of pathologic fracture of the femur resulting from malignant lesions, a metastasis from carcinoma of the breast and an osteogenic sarcoma, which were treated by intramedullary nail fixation are presented. Serial roentgenograms revealed that the entire shafts of the involved femora were replaced by tumor tissue during periods of eight and six months, respectively. Despite this, the femora

remained stable clinically and both patients remained mobile and relatively free from pain until shortly before death about eight months after occurrence of the fracture. Both patients received treatment with radioactive phosphorus and local roentgen irradiation with no apparent mechanical interference from the intramedullary rods, although treatment was apparently ineffective in retarding local spread of the lesions.—*Harold C. Hamilton, M.D.*

RAVELLI, A. Zum Röntgenbild des proximalen Schienbeindrittels. (The roentgen appearance of the proximal third of the tibia.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Jan., 1955, *82*, 48–52.

This article deals with the so-called companion shadow at the fibular side of the proximal third of the tibia. This shadow resembles a periostitis but it is well known that it is a normal bone detail without any pathological significance. The interpretation of this shadow varies as to its anatomical cause and the article considers the different interpretations, particularly as expressed in Köhler's and Grashey's well-known books.

There are drawings and reproductions of roentgenograms of anatomical specimens as well as of human legs. It is concluded that the shadow is produced by the "tuberositas tibiae."—*E. G. Wissing, M.D.*

SCHWABE, HASSO K. Tuberkulöse Veränderungen des Bandapparates der Wirbelsäule, zugleich ein Beitrag zum Infektionsmodus der Spondylitis tuberculosa beim Erwachsenen. (Tuberculous changes of the band apparatus of the spine; a contribution to the mode of infection of tuberculous spondylitis in adults.) *Tuberkulosearzt*, Dec., 1954, *8*, 747–754.

Formation of spurs and bridges in tuberculosis of the spine has been found in about 25 per cent of such cases, probably due to a specific ossifying periostitis and not due to a secondary spondylosis.

Two cases with active cavernous lung disease are described. There appeared to be a primary isolated tuberculosis of the band apparatus which seemed to have been the cause of clinical and roentgen findings. After many months of pain and clinical signs of radiculitis, without roentgenological abnormalities, localized periosteal bridges became evident without involvement of the bony structure of the vertebra.

Following surgical excision of the lung cavities, the clinical symptoms of spinal disease disappeared. These cases suggest to the author that a lymphogenous or hematogenous tuberculous infection of the band apparatus of the spine is possible and that surgical removal of the active lung focus arrested the tuberculosis of the spine. The only roentgen sign which remained was a localized intervertebral bridge.—*H. W. Hefke, M.D.*

WALKER, C. S. Calcification of intervertebral discs in children. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 601–605.

Most cases of calcification of the intervertebral disks have occurred in adults and have seldom been found in children. Only 7 cases have been fully recorded in the literature according to the author. Unlike calcification in adults, the condition can be accompanied by quite marked symptoms and signs.

The patient reported in this article was a girl, aged ten years, who had pain behind the left hip for ten weeks previous to admission to the hospital. She had some limitation of motion of the upper lumbar spine with tenderness to the left upper lumbar region. There was marked lumbar lordosis and some thoracic kyphosis. Roentgen examination showed dense areas of calcification in three disks, the lowest thoracic and upper two lumbar. The uppermost mass corresponded fairly accurately with the position of a normal nucleus pulposus but with slight bulging into the body on either side. The lowest calcified area was larger and was not only bulging into the posterior thirds of both adjacent bodies but was also projecting backwards into the intervertebral canal. The middle calcified mass appeared to be disintegrating and a large part of it had escaped laterally to the left of the disk.

After rest in bed for two weeks, a second roentgenogram showed that the lowest disk was protruding further into the canal and more of the calcified mass had "oozed" out laterally. The spine thereupon was immobilized, and examination after four months showed that the calcification between T12 and L1 had almost disappeared. Recent roentgenograms, approximately ten years after the child was first seen, revealed that very slight narrowing of the affected disks was present and apart from the slight persistance of the bulging into the posterior thirds of the bodies adjacent to the affected disks, the vertebrae had normal contours.

It is notable that, in this case, the calcified masses caused no symptoms while they were lying dormant. The only two occasions on which the patient complained of pain corresponded with the disintegration of the calcified zone before its absorption. The cause of calcification in intervertebral disks is uncertain.—*Ralph S. Bromer, M.D.*

VAN DE BERG, F., and CRÈVECOEUR, M. La disco-radiculographie lombaire avec produit de contraste hydrosoluble monoïodé. (Lumbar disco-radiculography by means of monoiodine hydrosoluble contrast medium.) *J. belge de radiol.*, 1954, *37*, 464–480.

The authors report the result of their study of the contour of the lower dural sac by using a 20 per cent solution of sodium salt of monoiodomethane sul-

fonate. Since this water soluble compound is absorbed within half an hour, it is important to proceed with the roentgenographic examination of the lumbar area rapidly after the intradural injection.

The proper preparation of the patient, his positioning (the roentgenographic table is inclined 25 degrees so as to permit accumulation of contrast medium in the lower dural sac by gravity) and the preliminary intradural injection of 1 cc. of 0.5 per cent nupercaine are stressed. The spinal anesthesia setting in, using the same intradural needle, which had remained in place, 10 cc. of 20 per cent sodium monoiodomethane sulfonate is injected very slowly. During the ten minutes after the end of the injection eight roentgenograms of the lower lumbar area are taken. The after care of the patient consists in seeing that he remains seated in his bed for twenty-four hours.

No serious complications have been observed by the authors. The nausea and general malaise, as well as the lower lumbar anesthetic effects, are all transient and of no importance. Severe shock and epileptiform cramps reported by some investigators are thought to be due to the horizontal position of the patient sometime during the first twenty-four hours, and pollution of the hydrosoluble contrast medium by free iodine.

The advantages of this method are clearer demonstration of the dural sac by herniation of discs or other pathology; the contours of the nerve roots are much better visualized than by the oily opaque media; and as the contrast medium is resorbed within half an hour, there is no subsequent danger of chronic arachnoiditis.

Several fine roentgenographic reproductions accompany the article.—*J. N. Sarian, M.D.*

Kloss, K. Zur Therapie der Myelographie-Zwischenfälle nach Verwendung wasserlöslicher Kontrastmittel. (Treatment of the complications of myelography following use of water soluble contrast media.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Jan., 1955, *82*, 86–90.

The complications accompanying use of water soluble contrast material for myelography are discussed and explained.

During 250 myelographies with 10 cc. Kontrast-U 20 per cent, the following side effects occurred: 6 cases of immediate pain and cramps, 2 cases of delayed pain and collapse, 1 case of epileptiform attack after eight hours and 30 cases of delayed reactions of headache, dizziness and aggravation of the original complaints.

The most annoying reactions are those of immediate pain and usually are due to incomplete lumbar anesthesia. The importance of meticulous technique in performance of this procedure is stressed. Lumbar puncture is done in the sitting position, which facilitates the exact placement of the needle; 10 cc. of pantocain is injected in the lateral decubitus position and mixing with the spinal fluid assured by repeated withdrawal and reinjection. Tilting of the table to more than 15–20 degrees is avoided. If pain occurs complete narcosis with pentothal is produced, after waiting one to two minutes for spontaneous disappearance of pain. Exposures can be made with the patient asleep.

Late complications can be avoided by placing the patient in half sitting up position for at least ten hours after myelography instead of placing him flat on his back. The few cases which still had pain or collapse after three to six hours responded readily to opiates and vasoconstricting drugs. Delayed pain responded to analgesics.

The number of complications with myelography with water soluble contrast media are few. Fatal cases reported could have been avoided by complete lumbar anesthesia and more vigorous treatment of collapse. There is no reason to abandon this method of examination.—*Frederick M. Reis, M.D.*

Lamb, Douglas W. Localized osteochondritis of the lumbar spine. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 591–596.

Osteochondritis of the lumbar spine (Scheuermann's disease) presents a characteristic and widely recognized clinical and roentgenographic picture. It usually produces symptoms between the ages of twelve and seventeen, and roentgenographic examination shows a minimal wedging of the bodies of adjacent vertebrae in the middle and lower regions of the thoracic spine. Considerable attention had been paid to this common condition but there is little reference in the literature to an affection, which appears to be of the same nature, involving a small localized area of the lumbar spine and producing a characteristic defect of the vertebral body. The purpose of this article is to report cases in which a similar localized condition was found in the lumbar spine and to emphasize the importance of differentiation from tuberculosis which it may resemble closely.

Seven cases of localized lesions of the lumbar spine in children are reported in the article and are considered to be examples of localized vertebral osteochondritis. All patients complained of pain, 6 in the lumbar area, the other in the region of the hip. In 3 there was considerable paravertebral muscle spasm with restriction of movement. The roentgenographic appearance was alike in each case: there was narrowing of the disk space with a characteristic defect of the upper or lower anterior corners of the adjacent vertebral bodies. In some a central disk protrusion was present.

It seems likely that this condition, according to the author, is caused primarily by weakness of the intervertebral disk, either congenital or posttraumatic. The constant narrowing of the disk space and the occasional appearance of Schmorl's nodes

are suggestive of this hypothesis. This also offers a possible explanation of the characteristic defect at the upper or lower anterior corners of the vertebral bodies bounding the disk space. As the fluid nuclear material escapes from the disk, with resultant narrowing of the space, it may break the cartilage plate to form Schmorl's nodes, but it may equally allow pressure anteriorly. The tough anterior longitudinal ligament resists this force which is directed on to the upper or lower corner of the vertebral body with consequent pressure necrosis of bone and formation of the roentgenographic defect described and illustrated in the article.

It is important to distinguish this condition from spinal tuberculosis. These cases have shown slow regeneration of the vertebral defect over a period of months.—*Ralph S. Bromer, M.D.*

ODELL, RICHARD T., and KEY, J. ALBERT. Lumbar disk syndrome caused by malignant tumors of bone. *J.A.M.A.*, Jan. 15, 1955, *157*, 213–216.

On the orthopedic service at Barnes Hospital, St. Louis, during the past twelve years, the authors have encountered only two tumors of the spinal cord or its appendages (an unsuspected ependymoma and a neurofibroma that was suspected) in a series of over 1,200 operations for intervertebral disk. During the same period they have seen 19 patients with malignant tumors involving the bone in this area; 10 had symptoms of an intervertebral lesion that led to their being treated conservatively with this diagnosis; 9 were operated on for an intervertebral disk.

In 11 instances nothing abnormal was noted in the roentgenograms although subsequent observation would indicate that macroscopic tumors were present in the bone. It is pointed out that a bone may be invaded or partially destroyed by tumor and yet exhibit no change in the roentgenogram.—*James J. McCort, M.D.*

WILLIAMS, ROHAN. Complete protrusion of a calcified nucleus pulposus in the thoracic spine. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 597–600.

The clinical and roentgenographic diagnosis of rupture of the intervertebral disk and backward prolapse of the nucleus pulposus is commonplace in the lumbar region and is sometimes made in the cervical region also. Calcification of the nucleus pulposus is occasionally observed roentgenologically in the thoracic spine, but rarely in other vertebral regions. Prolapse of the nucleus pulposus is distinctly uncommon in the thoracic spine. The author's case report is concerned with the demonstrable prolapse of a completely calcified nucleus pulposus in the lower thoracic spine—a rare occurrence.

The patient was a woman, aged forty-seven years, who had severe pain in the region of the left costal margin and the sternum. Roentgen examination revealed slight anterior marginal osteophytic lipping affecting the bodies of the fifth to the eighth thoracic vertebrae. In the lower thoracic spine between the bodies of the tenth and eleventh thoracic vertebrae, a calcified nucleus pulposus was shown, the uncorrected measurements being anteroposterior, 21 mm. and superoinferior, 7 mm. The transverse measurement was 7 mm. Anteriorly at the same level was some dense separate calcification, probably in the anterior margin of the annulus fibrosus. There was slight osteophytic lipping at the lower margin of the tenth and the upper margin of the body of the eleventh thoracic vertebrae.

Two months later, roentgenographic examination revealed that the whole of the calcified nucleus pulposus had been extruded posteriorly and was seen to lie behind the upper part of the body of the eleventh thoracic vertebra and was probably intact. The intervertebral disk space had become appreciably narrowed. The slightest remnants of the calcification of the annulus fibrosus near the site of the nucleus pulposus were just detectable. The anterior marginal calcification showed a slight forward protrusion. The posterosuperior margin of the body of the eleventh thoracic vertebra was defective, as if marginal avulsion had occurred when the complete disk rupture and nuclear protrusion had occurred.

The author states that there was roentgenographic evidence of the escape of the whole nucleus pulposus posteriorly. Judging from the history, there was pressure on the nerve root which was possibly relieved when the total rupture occurred. Close inspection of the roentgenograms and laminagrams suggested that pressure on the cord would have been almost inevitable if the posterosuperior margin of the body of the eleventh thoracic vertebra had not given way so that the nucleus pulposus did not bulge into the spinal canal to any marked extent, behind the plane of the posterior margin of the body.—*Ralph S. Bromer, M.D.*

DAHLIN, DAVID C., BESSE, BYRON E., JR., PUGH, DAVID G., and GHORMLEY, RALPH K. Aneurysmal bone cysts. *Radiology*, Jan., 1955, *64*, 56–65.

Aneurysmal bone cyst is a distinct clinical and pathologic entity. The authors report the clinical, roentgenologic and pathologic features of this disease found in 26 patients among 2,000 primary bone lesions reviewed at the Mayo Clinic.

The condition usually occurs in the younger age group, having been seen somewhat more often in females in this series. The lesions have been found in practically all bones except the mandible and facial bones. The majority of lesions occur in the vertebrae, or long bones.

Clinical complaints include pain, swelling, limitation of motion, and tenderness. The duration of symptoms is relatively short.

The roentgenologic manifestations vary. The typical lesion is characterized by a well circumscribed rarefied area, usually occupying an eccentric position in the bone, which shows either fine or coarse trabeculae, or both. The trabeculae are more coarse and more numerous toward the edges of the lesion, producing a characteristic soap-bubble or honeycombed appearance. The cortex may bulge eccentrically or be absent at the periphery of the affected segment of bone. Frequently, only irregular remnants remain. Small bones such as the fibula or rib may bulge symmetrically in a fusiform fashion. About the disrupted cortex there is a very thin shell of bone, representing periosteal new bone, around the periphery of the lesion. In long bones, the end of the shaft is the site of predilection. In growing bones the lesion is on the diaphyseal side of the epiphyseal cartilage. The tumor does not invade or affect the epiphyseal cartilage. In older patients, in whom the epiphyseal cartilage is no longer present, the epiphyseal portion of the bone is generally affected. The joint spaces do not become involved.

In the spinal column the lesion is usually limited to only a part of one vertebra. Trabeculae are usually more sparse than in long bones, but the thin outline of periosteal new bone is apparent.

The microscopic study of these lesions, particularly the atypical ones, is occasionally necessary for verification of diagnosis. Profuse bleeding at time of surgery is common. The gross and microscopic characteristics of this highly vascular lesion are described in detail. In general, there are various-sized engorged vascular spaces whose walls lack most of the features of normal blood vessels, the connective tissue septa often contain thin strips of osteoid (fiber bone), and there are pools of unclotted blood in the vascular spaces.

The roentgenologic differential diagnosis of aneurysmal bone cyst is discussed in relation to benign giant cell tumor, hemangioma, fibrous dysplasia, and simple bone cyst. Nine figures of typical roentgenograms, the gross and the microscopic appearance of these lesions are included.—*E. E. Seedorf, M.D.*

Kellsey, David C., and Sproat, Harry F. Echinococcus disease of bone; report of a case. *J. Bone & Joint Surg.*, Dec., 1954, *36-A*, 1241–1248.

Experience with echinococcus disease of bone in the United States has been extremely limited, because up to the present time only 10 cases have been reported. The patient described in this article although studied in the United States, apparently acquired the disease in Puerto Rico. In their study of the literature, the authors failed to find any reports which included as complete a pathological study as is their present report.

Man, although a relatively uncommon intermediary host, may be infected by the ova-containing excreta of dogs. This is more particularly seen in the great sheep-raising areas of the world. The initial bone lesion may remain localized or extend to an entire bone. Ordinarily bones are first involved by arterial spread after the embryos have cleared the first two filters—the liver and the lungs. There is an absence of both clinical and roentgenographic signs of hydatid infections of bone for a long period of time. The first stage of bone involvement consists in an invasion of the relatively well vascularized area of the epiphysis. The hydatid cysts then spread along lines of least resistance. Roentgenograms at this stage may reveal multiple minute, rounded, cystlike spaces limited by thin trabeculae. Early in the disease, little periosteal bone develops and there is little cortical expansion. However, as a result of prolonged and clinically almost silent irritation hyperostosis usually predominates over osteolysis. Later in the disease a second stage of bone involvement is noted. Enclosed cystic areas within the development of a surrounding inflammatory osteitis appear. There is never any sharp demarcation between the normal and the diseased areas, and this is a helpful fact in making a differential diagnosis. As the disease advances, the cortex finally becomes thinned by increasing internal pressure and pathological fractures may occur.

During the first stage of bone involvement, the disease may be mistaken for such rarefying bone lesions as osteosarcoma, giant-cell tumor, von Recklinghausen's disease (osteitis fibrosa cystica), vertebral angioma or monostotic Paget's disease. During the second stage of bone involvement, the disease may be confused with osseous tuberculosis. As the disease advances, progressive hyperostosis results and in its second stage the condition may thus become confused with tuberculosis or lues, but at this time clinical differentiation can be made. The authors suggest an adequate biopsy as the best solution to the problem of differential diagnosis.

In the case reported, roentgenograms revealed a thickening of the cortex in the left humerus and multiple areas of radiolucency. The entire humerus was involved. The skeletal survey was otherwise negative.

No drug therapy was administered during the patient's stay in the hospital, as no drugs now known are efficacious in this disease. As the patient was completely asymptomatic and the finding of osseous hydatidosis was an incidental one, the authors did not feel that they should urge him to undergo disarticulation at the shoulder, although this seemed to be the only solution if they hoped to prevent osseous spread of the echinococcus infection in the future. Approximately eleven months after discharge from the hospital a follow-up report stated that he still had no obvious systemic or local spread and was in good health.—*Ralph S. Bromer, M.D.*

BIERLING, G., and REISCH, D. Über das Sudecksche Syndrom nach Frakturen. (The Sudeck syndrome after fractures.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Jan., 1955, *82*, 1–14.

Most publications assert that the Sudeck syndrome is the most significant complication of bone injury, with the most severe instances of this disease being seen during the course of healing of fractures. The concept of the individual author varies widely as to when decalcification in the region of the fracture should be called the Sudeck syndrome or "physiological repair." The mere fact that one can recognize the decalcification on the roentgenogram places the condition as pathological.

The authors have studied all stages of the Sudeck syndrome in the hope of arriving at some conclusion regarding some interesting questions in respect to this disease.

Roentgen findings lag two to three weeks behind the clinical manifestations of the Sudeck syndrome. Yet the diagnosis usually is established on the basis of the roentgen examination. The diffuse decalcification in the first stage of the disease occurs in children under ten years of age. The first roentgen manifestation of the Sudeck syndrome varies with the localization of the fracture. In certain cases there is seen a band-like metaphyseal decalcification, while in others a band-like epiphyseal or spotty decalcification is noted.

A report is made of 3,170 patients with closed fractures of the extremities. Only those cases in which the known roentgen signs were seen were diagnosed as Sudeck's syndrome, 587 such cases, or 18.5 per cent, being so classified. When one excludes fractures of the phalanges, where this condition is seen very seldom, the percentage rises to 22. Of 1,917 fractures in the upper extremities, Sudeck's syndrome was seen in 13.5 per cent; of 1,253 lower extremity fractures in 26.3 per cent. The more static circulation in the lower extremities predisposes to post-traumatic dystrophy. Of the upper extremity fractures, those in the forearm constituted one-third of the Sudeck syndrome cases, mostly in young patients; in the lower extremity fractures, those around the knee joint comprised the largest number. The author's found that women in the fifth and sixth decades are the most prone to develop the Sudeck syndrome. The significance of endogenous factors could not be determined. Of 258 Sudeck's syndrome cases in the upper extremities, 38 were of the severe type and of 329 in the lower extremities, 70 were severe. The predisposition of the lower extremities is again demonstrated. The previous age group of the fifth and sixth decades also is preponderant.

The diffuse form of decalcification in Sudeck's syndrome occurs usually in the first decades, although it may occur less frequently up to the end of the second decade. The diagnosis may be very difficult and often roentgenography of the normal side is necessary for comparison. This form returns to normal very rapidly. Particularly in radial, and malleolar fractures, the first sign of Sudeck's syndrome is a band-like radiolucency in metaphysis, followed by subchondral and later by spotty decalcification. In the elbow and knee regions, there is first a subchondral decalcification. In fractures of the shafts of the long bones there is a "mouse-eaten" type of bone change similar to that seen in tumors.

The Sudeck syndrome was seen rarely after hip-nailing. In calcaneal fractures, with an extension pin in that bone, the condition was noted almost regularly, but when such a pin was not inserted, the syndrome was present more seldom.

Many forms of therapy including nerve block, diathermy, etc., had been used in Sudeck's syndrome. The authors state that many patients with severe form of this condition have recovered spontaneously. Since androgen has been of value in postmenopausal osteoporosis, the author's used this hormone in patients with Sudeck's syndrome, especially since this condition is a frequent complication after fractures of the extremities in the climacteric and post-climacteric individuals. Nineteen patients were given injections of 50 mg. doses of methyl-androstendiol two to three times a week for a total dose of 750 to 1,000 mg. Particularly in those patients in the fifth and sixth decades, the soft tissue swellings regressed after the first two injections, and the pain in the region of the fracture improved or disappeared. In certain individuals, this improvement was temporary, even though the treatment was not completed. No change on the roentgen films could be noted after six to eight weeks.

Forty patients were given roentgen therapy over the sympathetic chain and the affected extremity, using 180 kv., 0.5 mm. Cu filter, 30 cm. distance, with doses of 100 roentgens every third day. In the cervical spine region, two paravertebral fields were used, and usually one area over the affected extremity. The thoracic and lumbar spine regions were divided into three fields. After all the fields were treated, a rest interval of ten to fourteen days was given and the series was repeated. The authors believe that pain was relieved by the above treatment. At present, they combine androgen with roentgen therapy, since the latter works more slowly while the former produces a more rapid but temporary effect. —*Samuel Richman, M.D.*

LIESS, G. Multiple symmetrische Umbauzonen (Milkman-Syndrom) ungewöhnlicher Ätiologie und Lokalisation. (Multiple symmetrical repair zones [Milkman's syndrome] of uncommon etiology and localization.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Jan., 1955, *82*, 15–27.

The manner in which bone repair secondary to

trauma is manifested depends on the disproportion between the injury and the ability of the bone to withstand such damage. Thus, a march fracture represents a relatively acute onset of bone insufficiency. Histologically, the repair zone is characterized by the break down of old bone and simultaneous formation of slightly differentiated fibrochondro-osteoid connective tissue. The roentgen translucent area represents not a break in continuity, but a replacement of the bone by noncalcified tissue. In osteomalacia, the repair zone of newly formed osteoid tissue remains uncalcified for a long time.

Since Milkman in 1943 described a case of multiple symmetrical bone repair zones refractory to therapy, numerous similar reports have appeared in the literature. Patients with osteomalacia secondary to vitamin D deficiency or exhibiting an unfavorable course as a result of a specific disease should not be so classified. Since the histological status is similar in all these cases, a roentgenological differentiation as to the etiology of the osteomalacia cannot be made. This can be done only clinically and on the basis of subtile metabolic studies.

A thirty-one year old female with neurofibromatosis of the skin showed generalized marked osteoporosis. Over 30 repair zones, primarily symmetrically distributed over the entire skeleton were found. Usually, a transverse line of decreased density through the entire bone was noted. Occasionally, faint periosteal proliferation could be seen. Iliac bone biopsy was diagnosed as osteomalacia. Blood calcium was 8.6–9 mg. per cent; inorganic serum phosphorus was lowered to 1.8 mg. per cent; alkaline phosphatase was elevated to 14.8 King-Armstrong units; calcium excretion in stool and urine was increased. After treatment with ultraviolet rays, high doses of vitamin D, and supplementary phosphorus and calcium, clinical, metabolic, and roentgenological improvement was noted. It was concluded that a disturbance in the metabolism of calcium and phosphorus was the underlying factor in the osteoporosis of this patient. Reports in the literature estimate the frequency of bone changes in neurofibromatosis from 7 to 40 per cent. Osteomalacia of the entire skeleton has been mentioned relatively seldom. The relationship between the osteomalacia and neurofibromatosis is difficult to evaluate. The possibility of a resistance of the osseous tissue to vitamin D is to be considered. The localization of the "insufficiency damage" is determined on the one hand by the resistance to trauma of these areas of the bones and on the other hand by the dynamic (muscular) and static strength to absolute or relative continuous injury. The constant localization of the repair zones over the entire skeleton with preponderance in the pelvis and upper extremities, is of great differential diagnostic value in osteomalacia. A variation from this localization is seen when there is bowing of the bones, since the repair zones are formed first in bones which are bowed.

A third variation of location of the repair zones was seen in the following two cases of osteomalacia: *Case 1.* A forty-five year old man first noted pains in pelvis and lower extremities two months after falling on his back. His stature decreased and he used two canes for walking. Seven years after the injury he was diagnosed as having sustained multiple fractures and was confined to bed rest. Roentgen studies two years later showed multiple repair zones with marked degree of generalized osteomalacia. In addition, transverse bands of decreased density in the region of the epiphyses of the radii, distal and proximal ends of the humeri, in the symphysis pubis, and in the metacarpals, were made out. These simulated the repair zones seen in Sudeck's syndrome. Spontaneous fractures were noted in some of the atypically situated repair zones. Blood chemistry showed: calcium 8.2 mg. per cent, phosphorus 1.5 mg. per cent, and low serum alkaline phosphatase. Treatment with high doses of vitamin D yielded no improvement after four and one-half months. *Case 2.* A thirty-three year old female who was well until 1944 began to notice pains in her back, particularly when climbing steps. Marked osteoporosis of all the bones developed. As in the preceding case, transverse bands of decreased density were noted in the epiphyseal regions. These bands extended beyond the epiphysis and almost completely obliterated the outline of the heads of the femora, humeri, and radii. In these atypically located repair zones, spontaneous fractures and dislocations were present. Biopsy from one of these areas showed osteomalacia. Blood phosphorus was near 1 mg. per cent, alkaline phosphatase was elevated slightly, while the calcium approached the lower limits of normal.

The last 2 cases were classified under renal acidosis with tubular insufficiency. The cause of the atypical location of the repair zones in these two patients may be allied closely to the changes (epiphyseal bands) seen in Sudeck's syndrome.—*Samuel Richman, M.D.*

Singer, Martin. Fanconi's syndrome; treatment by massive dosage of calciferol and two-stage osteotomy. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 632–636.

The author states that rickets caused by deficiency of vitamin D has almost disappeared in Great Britain, and for this reason attention is being focused more and more on the group of conditions which in the past has been termed "resistant" and "renal" rickets. In renal glomerular rickets, which is in fact the classical "renal rickets," both glomeruli and tubules are damaged or destroyed, leading to retention of urea, phosphates, and other substances normally excreted in the urine. In renal tubular rickets, there is an inherited or acquired defect of tubular reabsorption. Various functions may be lost, depending on the site and extent of the defect. For clinical purposes this group can be subdivided into

six types, according to the nature of the tubular reabsorption defect: (1) phosphates only (low blood phosphorus); (2) phosphate plus glucose (glycosuria); (3) phosphate plus glucose plus amino-acids (amino-aciduria, including cystinuria); (4) phosphate plus glucose plus amino-acids, with faulty urine acidification (low CO_2 combining power); (5) phosphate, with faulty urine acidification and high urinary calcium content; (6) phosphate, with faulty urine acidification, defective ammonia formation, and high urinary calcium content.

The case reported in this article accorded with Fanconi's syndrome (type 3 or 4 of the classification) the signs of which are considered by Dent (1952) to be due to an inherited defect of reabsorption affecting primarily the proximal tubules of the kidney and involving amino-acids, sugar and phosphates. In this case the roentgenograms were typical of severe rickets.

The patient was treated for nearly a year with calciferol daily until he was two and one-half years of age, dosage of 100,000 units. Thereafter the dosage was stepped up to 500,000 units daily. No citric-acid-sodium-citrate solution was given. Healing of the rachitic lesions followed and there was simultaneous improvement in the biochemical state. The amino-aciduria disappeared. Two-stage osteotomies were undertaken for the correction of severe bowing of the tibiae. No toxic symptoms have as yet developed from the calciferol therapy.—*Ralph S. Bromer, M.D.*

GERSTEN, A., and LECOMTE-RAMIOUL, S. Etude radiologique des jonctions chondro-costales dans le rachitisme. (Roentgenological study of the costochondral junctions in rickets.) *J. belge de radiol.*, 1954, *37*, 454–463.

In infants under fifteen months of age, the early stage of rickets is diagnosed by biochemical tests with maximum certainty.

In the experience of the authors, roentgenographic findings at costochondral junctions and at the wrists are also of definite importance in diagnosing rickets in its initial stages. These findings are enlargement of the distal end of the ribs, scalloping of the epiphyseal lines and at times irregularly serrated configuration at the costochondral junction. This description is also true at the level of the distal radial epiphysis. Only in scurvy and certain types of leukemia can similar roentgenographic changes be encountered. However, in scurvy there is the characteristic circling of the epiphyseal centers at wrist and at knee whereas in leukemias the alterations of the epiphyseal lines are accompanied by marked generalized osteoporosis.—*J. N. Sarian, M.D.*

DECHAUME, M., ALBEAUX-FERNET, M., GELINET, M., and PAYEN, J. A propos d'une observation de syndrome d'Albright. (Concerning an observation of the Albright syndrome.) *Presse méd.*, Dec., 1954, *62*, 1787–1789.

Albright's syndrome or polyostotic fibrous dysplasia is a systemic bone disease which occurs predominantly in females and develops during puberty and adolescence. Its relationship to specific endocrine dysfunctions has not been definitely established.

The authors report the rare case of a male, Mediterranean, aged twenty-four, with clinical manifestations of the disease dating back at least fifteen years. There was no evidence of precocious puberty. At the age of five the patient had suffered a greenstick fracture of the right proximal femur. Four years later the right lower extremity became markedly shortened, and roentgenograms at that time showed marked upward curving of the femoral shaft above the level of the hip. The head and jaw bones became markedly enlarged, and there was frontal and occipital bossing and spreading apart of the orbits with convergent strabismus of the eyes.

Roentgenograms at the age of twenty-four years revealed marked enlargement of the cranial bones and of the maxilla with far advanced fibrocystic changes which were predominantly hyperostotic and osteoplastic in character. The mandible also was markedly enlarged but showed only fibrocystic changes with osteoporotic character. Typical expansile cystic lesions were found in the tubular extremity bones, involving diaphyseal portions as well as metaphyseal and epiphyseal areas. There were no typical cafe-au-lait spots, but some abnormal skin pigmentation was noted in both gluteal areas.

Blood chemistry findings and other laboratory studies were negative, including specific hormonal studies. Only the sugar tolerance and the insulin tests suggested diminished pituitary function.

The article contains reproductions of six typical roentgenograms, photographs, microphotographs, curves and graphic illustrations.—*Ernest Kraft, M.D.*

CAMERON, J. A. P., YOUNG, W. B., and SISSONS, H. A. Metaphysial dysostosis. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 622–629.

In 1934 Murk Jansen described an unusual case of skeletal growth disturbance under the title of "Metaphysial Dysostosis." The case presented in this article is reported as an example of metaphysial dysostosis in which no evidence of a relationship with renal disease could be found on careful study, including clinical, roentgenological, biochemical and biopsy investigation. The case appeared to be the only classical example of metaphysial dysostosis that has been described since Jansen's report in 1934 and for this reason the authors put it on record.

The patient in this case report is a Chinese boy, aged seven years, who was seen on account of de-

formity of both lower limbs. On physical examination he also had some deformity of the chest and upper limbs. The fingers were "clubbed" and the ribs showed a rosary at the costochondral junctions. The lower limbs revealed marked genu valgum with anterior bowing of the femora, rotation of the tibiae and angulation of the lower ends of the tibiae. In the roentgenograms, the ends of the long bones were expanded to a greater or lesser degree as if the process of remodelling had failed. In some bones this expansion was very pronounced and the bone had the shape of a club. The expanded ends were cupped, the amount varying in different bones, but in some cases being sufficient to enclose the greater part of the epiphysial bone center. The walls of the cup were formed of sclerotic thickened bone and in some bones appeared to be fragmented or possibly fractured. The bottom of the cup was formed by a zone of fairly dense irregular interlacing trabeculae, which seemed to be compressed so that an area of increased density demarcated the abnormal bone from bone of apparently normal structure in the diaphysis. The cupped expanded ends of the bones appeared to contain a material of soft tissue density in which scattered irregular areas of density were seen which in some bones resembled isolated fragments of dense bone; in others amorphous calcification; and in others punctate calcification. In the lower end of the radius and ulna there was a mosaic pattern of soft-tissue densities separated either by irregular transradiant lines or lines of increased density. The femora were bowed anteriorly to a marked degree. The tibiae and fibulae also appeared to be bowed, but the deformities were caused not so much by bowing of the shafts as by angulation at the junction of the disordered hyperplastic mataphysis and the shaft. At sites where angulation had occurred there was increased bone density most marked on the concave side. The shafts of the long bones were thicker than normal, but the cortices were not particularly thickened, except at sites of bowing where there was definite buttressing, as in the tibiae where cortical buttressing extended all along the posterior and medial parts of the shafts. The distal bones of the limbs appeared to be a little short relative to the length of the proximal bones and the fibulae appeared short in relation to the tibiae. The small long bones of the hands and feet were shorter and broader than normal so that the hands had a stumpy trident appearance and the feet appeared wide and short. They exhibited metaphysial changes similar to those occurring in the long shaft bones and were interesting in that, although in the distal and middle phalanges cupping and loss of density were the most marked features, in the proximal phalanges islands of slight density could be seen within the cups, and in the metacarpals these islands appeared still denser as if bone had been laid down within them.

The anterior ends of the ribs and the inner ends of the clavicles were expanded and cupped and showed changes similar to those described in the long bones. In the pelvis the acetabula were deformed and enlarged and there was a marked irregularity of their bony margins which seemed to be composed of dense nodules of bone and thickened interlacing trabeculae intermingled with clear areas. The skull was large and brachycephalic. The development of epiphysial and other centers of ossification in the hands and feet was markedly retarded. Although the outline of the pituitary fossa could not be seen in the roentgenograms of the skull, no evidence of pituitary disorder was present.

On the basis of the study of their case and of a comparison of it with the literature, the authors regard "metaphysial dysostosis" as a distinct entity among the chondrodystrophies (in which group are included achondroplasia, dyschondroplasia, diaphysial aclasis, dysplasia epiphysialis multiplex, dysplasia epiphysialis punctata, Morquio-Brailsford disease and gargoylism) and present their case as an example of this condition.—*Ralph S. Bromer, M.D.*

Jackson, W. P. U., Albright, Fuller, Drewry, Garth, Hanelin, Joseph, and Rubin, Mitchell I. Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. I. Familial metaphyseal dysplasia and craniometaphyseal dysplasia; their relation to leontiasis ossea and osteopetrosis; disorders of "bone remodeling." *A.M.A. Arch. Int. Med.*, Dec., 1954, *94*, 871–885.

This is a rare condition consisting of symmetrical expansion of one or both of the metaphyses of the long bones. In the metacarpals and phalanges the lesions are located only at the end where there is an epiphysis. The femora show an "Erlenmeyer flask" contour. There may be anterior bowing of the femora and an "S" bend in the tibiae; the legs may be long. Bone age is not affected. The cortex is thinned and the bone poorly mineralized so the involved bone is of homogeneous appearance.

Three cases are reported with excellent illustrations. The pathologic examination in 1 case showed the cortical bone to be moderately thinned and the spongy bone normal. All the bone was of the mature type and no abnormal osteoblastic or osteoclastic activity was present.

Five cases of craniometaphyseal dysplasia were collected from the literature which mistakenly were called osteopetrosis or leontiasis ossea. They had findings in the shafts as described above as well as sclerosis and overgrowth of the skull bones; the changes are sufficiently characteristic to be called craniometaphyseal dysplasia. One of these is reviewed in detail and a new case reported.

The differential diagnosis includes the following lesions:

1. Osteopetrosis, which shows an increased density

of all the bones of the body except the mandible. The bones are enlarged, but never to such an extent in the face as to cause leontiasis. Although the shape of the long bones may be similar to that in metaphyseal dysplasia, the marrow cavity is much reduced and there are longitudinal striations in the shafts. A further differential point is that the bones are weak and easily fractured. The patients have anemia and hepatosplenomegaly in the more severe cases. The defect in osteopetrosis is inability to resorb bone once it is laid down while in metaphyseal dysplasia only the cortical bone fails to be remodeled and the defect is local.

2. Leontiasis ossea, which may be due to Paget's disease, fibrous dysplasia or other causes. Some cases are associated with metaphyseal dysplasia.

3. Gaucher's disease and Cooley's anemia, which show similar bone contours but are differentiated by associated findings.

4. Vitamin A deficiency, which experimentally shows a few changes similar to those of metaphyseal dysplasia but no association has been found clinically.

Apparently there are "connecting links" and subvarieties between familial metaphyseal dysplasia, craniometaphyseal dysplasia, osteopetrosis and leontiasis ossea. The genetics are briefly considered.—*Martha Mottram, M.D.*

Jackson, W. P. U., Hanelin, Joseph, and Albright, Fuller. Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. II. Multiple epiphyseal dysplasia; its relation to other disorders of epiphyseal development. *A.M.A. Arch. Int. Med.*, Dec., 1954, *94*, 886–901.

In this condition there is delayed appearance of the epiphyses and they show irregularity in form and structure. The changes are bilateral and symmetrical and slowly progress to consolidation and late epiphyseal union. In adults the abnormal shape or joint angulation is the only residuum of the disease. The spine is usually normal but the epiphyseal plates may be irregular and the bodies reduced in height.

The patients have no clinical symptoms. There may be genu valgum or coxa vara, the hands are short and stubby, and the patients are short, usually around five feet. A biopsy is reported by Fairbank to show "cartilage irregularly placed, definitely hyaline and perhaps softer and more mucinous than normal."

Six cases with roentgenographic reproductions are reported. They all had the typical findings except that 2 patients showed spinal changes resembling those of juvenile epiphysitis. Two patients also showed continued non-union of fragments of the upper femoral epiphyses; this may be expected to cause a traumatic arthritis eventually.

Most cases reported are sporadic, although there is evidence of a dominant genetic trait.

The most important lesions from the point of view of differential diagnosis are:

1. Juvenile osteochondritis. The roentgen appearance of a single joint in multiple epiphyseal dysplasia may be indistinguishable from that of osteochondritis. The latter usually affects only a single center and arises in a previously normal epiphysis. There is always pain and the lesion may progress under observation.

2. Cretinism. In this condition there may be irregularity and fragmentation of the epiphyses which appear late. The centers are all late in fusing. The spine may be involved. There is delayed transformation of cartilage into bone.

3. Morquio's disease. In this condition the spine shows the most marked changes with kyphoscoliosis, flat thin irregular bodies with anterior central "tongue-like" projections. The disks are wide. The epiphyses appear late and are irregular and fragmented. The metaphyses are flared. The clinical picture is typical.

4. Gargoylism. Although there is some irregularity and distortion of the epiphyses in gargoylism, there is less fragmentation than in the conditions noted above. The vertebrae are short and rounded with irregular anterior surfaces. There may be an acute kyphosis with backward dislocation of a small wedged body.

5. Punctate epiphyseal dysplasia. In this condition there is premature epiphyseal calcification with punctate calcium deposits which may also occur in the synovia and tracheal rings. Most patients die in their first year.—*Martha Mottram, M.D.*

Jackson, W. P. U., Hanelin, Joseph, and Albright, Fuller. Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia, and related conditions. III. Progressive diaphyseal dysplasia. *A.M.A. Arch. Int. Med.*, Dec., 1954, *94*, 902–910.

In progressive diaphyseal dysplasia there is a symmetrical fusiform expansion of the mid-shafts (occasionally there may be two foci in one long bone). Two cases previously reported by Neubauer are reviewed; both showed progression of the lesions. Biopsy in these cases revealed a thickened periosteum with osteoblastic and osteoclastic activity. The cortical bone exhibited overgrowth and was transformed into cancellous bone. The marrow contained loose fibrous tissue.

The chief characteristics of progressive diaphyseal dysplasia are: the bone in the involved regions is irregular in density with loss of trabecular structure; the new bone is endosteal and periosteal; the skull may show thickening in the base or vault; the hands, feet, spine and pelvis have never been reported in-

volved; the epiphyses and bone age are normal; and the lesions are slowly progressive.

In the differential diagnosis one should consider:

1. Chronic sclerosing osteomyelitis, but a bone survey will clarify the diagnosis.

2. Infantile cortical hyperostosis. In this condition there is always involvement of the mandible and clavicle. The changes in the other bones may be asymmetrical.

3. Healing scurvey and rickets.

4. Hypervitaminosis A; both 3 and 4 show no essential change in bony texture and systemic findings are usually present.

5. Luetic periostitis, which is very similar in appearance to diaphyseal dysplasia and could cause confusion if there were extensive involvement.

6. Pulmonary osteoarthropathy; this shows soft tissue changes in addition to skeletal lesions and the hands and feet show major deformities.

7. Melorheostosis, which shows irregular densities of the cortex of a single bone.

8. Paget's disease, which may cause changes very similar to diaphyseal dysplasia but may be asymmetrical, and starts in one end of the shaft.—*Martha Mottram, M.D.*

De Pape, A. J. Multiple pseudo-cystic tuberculosis of bone. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 637–641.

The term multiple pseudo-cystic tuberculosis of bone was suggested in 1952 by Komins to avoid existing confusion in the nomenclature of certain sarcoid lesions of bone and some forms of osseous tuberculosis. It applies to characteristic multiple cyst-like bone lesions seen in tuberculosis in children. It differentiates these lesions from those due to sarcoidosis which Jüngling (1920) had grouped under the term osteitis multiplex tuberculosa cystica and, also, from the multiple osseous tuberculous lesions seen in adults and to which the term disseminated bone tuberculosis has recently been applied by Alexander and Mansuy (1950). Komins suggested that the term osteitis multiplex tuberculosa cystica (or cystoides) be discarded but that a modification, osteitis multiplex cystoides sarcoidosa, be retained for the osseous lesions of sarcoidiosis. Multiple pseudo-cystic tuberculosis of bone is uncommon.

The patient reported in this article is a boy, of North American Indian parents, who was ten months old when he died. The case report is of interest for three reasons: (1) the extensive involvement of the bones, particularly the skull; (2) the presence of a bone lesion at birth; and (3) the possible, though perhaps not likely, coexistence of syphilis. The diagnosis of tuberculosis was made on the basis of a strongly positive Mantoux reaction (1:1,000), roentgenograms showing extensive bilateral pulmonary changes suggestive of tuberculosis, the presence of acid-fast bacilli morphologically identical with *Mycobacterium tuberculosis* on smear and culture in pus obtained from the swelling on the forehead and a biopsy of a cervical lymph node showing necrosis, Langhans' cells and a histology compatible with a diagnosis of tuberculosis.

The Wassermann reaction was negative on two occasions, but a single Kahn test was weakly positive (1:8 dilution). It was felt that congenital syphilis could not be excluded, particularly in view of the family history of syphilis and some of the clinical findings. A three weeks course of penicillin was therefore given. The child showed improvement in appetite, was much less irritable and the nasal charge disappeared, but there was no appreciable gain in weight.

The roentgenograms of the patient revealed multiple cystic lesions in each radius and in some of the metacarpals, and the tibiae and fibulae. Somewhat circular areas of bone absorption were present in the skull. In the lung fields, paratracheal lymphadenopathy with diffuse parenchymatous lesions was considered evidence of tuberculosis.

Swelling of the fifth metacarpal of the right hand was present at birth. A roentgenogram of this hand at the age of seven months showed a cystic lesion suggestive of tuberculosis in this metacarpal. Histological examination at necropsy, three months later, showed focal diaphyseal necrosis, partial erosion and expansion of the cortex and thickening of the periosteum. No Langhans' giant cells but abundant acid-fast bacilli were present. It was impossible to say whether the initial lesion was tuberculous or whether tuberculosis had been superimposed on another condition such as syphilis. The author feels that the possibility that the child had syphilis cannot be ruled out but there is no doubt that the lesions seen on admission and at necropsy were all typically tuberculosus, with marked evidence of caseation and suppuration with many draining sinuses. According to Kampmeier suppuration with sinus formation is never seen in syphilis.—*Ralph S. Bromer, M.D.*

Potter, C. M. C. Osteomyelitis of the newborn. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 578–583.

Recent published reports of neonatal osteomyelitis in long bones are reviewed in this article and 6 cases in addition are described. The source of infection was usually the skin or the umbilicus and the common organisms were the hemolytic streptococcus and the *Staphylococcus aureus*. The long bones were affected. Of the 6 cases there were multiple foci of infection in 3, and all of the patients recovered. In only 1 case was there any serious constitutional upset. The other 5 fell into the group described previously as" benign." In 2 of the cases there was evidence of joint infection with complete destruction in 1 but with probably recovery in the other. Three patients formed sequestra which is unusual in infantile

osteomyelitis. In all cases rapid healing followed simple drainage, without interference with the bone itself, except in the 2 cases in which sequestra had first to be removed. A striking feature was the formation of abundant new bone beneath the periosteum. That this is so seldom associated with sequestration is possibly explained, by Green, by the early decompression permitted by the spongy nature of the infant's new bone and the loose attachment of the periosteum. The most important complication is suppurative arthritis which may lead to total destruction of a joint.—*Ralph S. Bromer, M.D.*

SNAPPER, I., SEELY, R., FALK, S., and FEDER, I. Osteomalacia in New York. *Ann. Int. Med.*, Nov., 1954, *41*, 893–909.

Osteomalacia is a metabolic disease of bone, that is brought about by insufficient amount of calcium and/or phosphorus in the blood. The most common cause is avitaminosis D, due to poor absorption from the intestine or failure to be exposed to enough sunlight and low intake of vitamin D.

There is failure of calcification of the bone matrix due to insufficient amount of calcium and phosphorus in the blood. Bone is continually being resorbed and laid down. If there is insufficient amount of calcium and phosphorus present, the bone becomes radiolucent and weakened which may end up in fractures. In compact bone these changes occur along the cement lines of the haversian canals. In cancellous bone it occurs at the edges of trabeculae.

The serum calcium for and/or inorganic phosphorus are low with very little calcium in urine. Later on in the disease the bones are exposed to more stress with an increased astroblastic activity and an increase in serum alkaline phosphatase. Early in the disease the alkaline phosphatase may be normal. The serum calcium and phosphorus are controlled by the parathyroid gland. If the serum calcium is low there is an increased secretion of parathormone, with resorption of the calcium from the matrix producing a normal serum calcium and an increased excretion of phosphorus in the urine producing a low serum phosphatase. At times there may be only a partial response from the parathyroid gland.

The authors present 4 cases of osteomalacia that was caused by fatty diarrhea. The fat soluble vitamin D was not being absorbed from the intestine due to the steatorrhea. The patients were treated with parenteral vitamin D and ultraviolet light.—*John Watts, M.D.*

ENGFELDT, B., ENGSTRÖM, A., and ZETTERSTRÖM, R. Biophysical studies of the bone tissue in osteogenesis imperfecta. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 654–661.

Osteogenesis imperfecta is a rare hereditary or sporadically occurring disease, primarily affecting bone but sometimes also involving other mesenchymal tissues. The bones are abnormally fragile, and multiple fractures of long bones often occur. The normal compact corticalis is absent and a loosely arranged spongy bone is formed. The etiology is unknown. The osteoblasts seem to be retarded in their function because of lack of differentiation. They are not capable of forming normal compact bone. Several investigations have been carried out to determine whether there is an abnormality in the metabolism of calcification. The blood chemistry, however, seems to be within normal limits at all ages.

In order to obtain further information about the disease the authors examined bone specimens from 4 cases by biophysical methods. The distribution of bone salts in different structures was studied with microradiography. The nature and the orientation of the mineral salts were studied with roentgen-ray diffraction. The distribution and orientation of collagen were studied in decalcified sections with polarized light.

Their results seemed to indicate that fragility of the skeleton is dependent not only upon the absence of normal compact bone but on the irregular arrangement of the organic matrix. Rebuilding of bone tissue was shown to occur in an abnormal manner. The mineralization of the bone was more uniform than is found in normal bone. The collagen had an abnormal organization and was sparse. The ultrastructure of bone salts and their orientation were as in normal bone.—*Ralph S. Bromer, M.D.*

JANES, JOSEPH M., HIGGINS, GEORGE M., and HERRICK, J. F. Beryllium-induced osteogenic sarcoma in rabbits. *J. Bone & Joint Surg.*, Nov., 1954, *36-B*, 543–552.

The original purpose of the study reported in this article was the production by chemical means of osteogenic sarcomas that would serve as test objects suitable for the application of ultrasonic energy. During an extensive experimental study of ultrasound the authors learned that such energy can inhibit the growth of bone in animals. Ultrasound is a term used to describe those frequencies in the acoustic spectrum which are beyond the range of the human ear. The use of ultrasound presented itself as a potential therapeutic procedure. As the authors proceeded with the work of producing and studying the tumors certain pathological changes were observed, particularly in the spleen, which seemed of sufficient interest to justify a separate report.

In 5 of the 10 animals injected with beryllium in the form of "zinc beryllium silicate" osteogenic sarcomas developed several months later. There was new bone formation in the medullary cavities of the long bones before malignant changes were apparent. There was atrophy of the spleen in those animals in which bone tumors developed, whereas the spleen seemed to be quite normal in those animals which

did not develop bone tumors. The tumors usually developed in the metaphyseal regions. More than one tumor often developed in the same animal. Heretofore, the authors state, little consideration has been given to a potential relationship between atrophic or aplastic changes in the spleen and development of tumors in the bones.—*Ralph S. Bromer, M.D.*

Blood and Lymph System

Fiandra, Orestes; Cervino, José Mannel; Navarro, Alfredo; Maggiolo, Jorge, and Mussio Fournier, J.-C. Dilatation jugulaire non obstructive. (Nonobstructive dilatation of the jugular vein.) *Presse méd.*, Dec. 25, 1954, *62*, 1826–1827.

The authors observed unilateral dilatation of the jugular vein in women at menopause almost exclusively on the left side. The dilatation varied with effort and position; symptoms were vague, often absent.

Blood pressure in the dilated vein was normal.

Phlebograms obtained by injection of 50 per cent diodrast into the left cubital vein showed a reflux of contrast material into the left jugular vein and a delay in flow at an angulation in the left brachiocephalic trunk.

No conclusion was drawn by the authors from this observation.—*Karol E. Matzinger, M.D.*

Petrie, E. A. Aberrant right subclavian artery. *J. Canad. A. Radiologists*, Dec., 1954, *5*, 46–51.

An aberrant right subclavian artery may be asymptomatic or may produce severe symptoms. The severity varies according to the degree of compression caused by the anomalous vessel on neighboring structures, especially the esophagus and trachea. Symptoms vary from mild dysphagia to marked nutritional deficiency.

An aberrant right subclavian artery can no longer be considered rare. The author reports 5 additional cases found in the living by roentgen methods from 1950 to 1952. All of these were recognized during fluoroscopy.

Irregularities in the development of the major branches of the arch of the aorta are numerous. In this anomaly, the right subclavian artery takes origin from the extreme left side of the aortic arch beyond the origin of the left subclavian artery. Consequently to reach the right side to supply the right arm with blood, it must cross the midline. In so doing it usually runs between the esophagus and the spine. This provides the means by which the diagnosis may be made by the roentgenologist in the living by the characteristic pressure defect on the esophagus posteriorly. Occasionally the anomalous vessel crosses anterior to the esophagus and rarely even anterior to both esophagus and trachea.

Since the site of origin is lower than it normally would be, the aberrant right subclavian artery has to pass upward as well as across, producing a characteristic spiral groove. This crossing may occur anywhere from the level of the sixth cervical vertebra to the fourth thoracic vertebra.

A classification and history of aortic arch anomalies is given in considerable detail.

The compression symptoms include paroxysmal or constant dysphagia, dyspnea (stridor, cough and hoarseness), cyanosis, retraction of the head and sometimes pain in the upper thoracic segments and the arms. These symptoms are all characteristically exaggerated during feedings and attempts at swallowing.

Other symptoms have also been attributed to aberrant right subclavian artery. Such changes as irregularity of radial pulse, enlargement of thoracic duct and symptoms like those of the scalenus anticus syndrome might be produced by aberrant right subclavian artery.

Fluoroscopy or roentgenography alone is quite sufficient to establish the diagnosis. Tracheobronchography may give added information but is usually not necessary. Angiocardiography may be of value.

Further diagnostic proof of the exact nature of the characteristic pressure defect in the esophagus is offered by use of a radiopaque catheter. The aberrant right subclavian artery is demonstrated by the introduction through the brachial artery of a radiopaque catheter. This method is less dangerous than angiocardiography. The thin catheter is seen lying along the oblique furrow in the esophagus caused by the aberrant right subclavian artery. This proves beyond any question the exact cause and nature of the characteristic roentgen findings.

In making a differential diagnosis the esophageal defect produced by a posterior type of right aortic arch, might be confused with that caused by the anomalous right subclavian artery. There are important points of differentiation. The posterior type of right aortic arch usually displaces the esophagus to the left as well as anteriorly. In the case of abnormal right subclavian artery, one can see a normal aortic arch slightly to the left of the vertebral column, usually not at the site of the esophagel defect. In aberrant right subclavian artery the defect is usually just above the arch but may be lower. It is smaller than the posterior defect produced by a posterior right aortic arch. In all these cases actual anterior displacement of the esophagus is less than that usually seen in posterior right aortic arch and the defect is smaller. The typical oblique defect produced by an aberrant right subclavian artery is readily seen in the anteroposterior projection if the barium is not too dense and if the amount swallowed is not too large. In the right anterior oblique projection the defect is seen to be on the posterior wall of the esophagus, when the artery passes posteriorly. The defect is anterior to the esophagus if the aberrant artery goes

between the trachea and esophagus. It is to be noted that a double aortic arch may produce somewhat similar findings. The latter may result in constriction of both trachea and esophagus. This combination of posterior esophageal compression and an anterior tracheal defect (with the absence of a demonstrable mediastinal mass) is almost certain proof of some sort of an encircling vascular ring.

Treatment of aberrant right subclavian artery is surgical when symptoms are severe or persistent. The dysphagia is abolished by severing the artery and removing it from behind the esophagus. This can be done without serious impairment of the blood flow to the arm, due to good collateral circulation.—*Stephen N. Tager, M.D.*

SZILAGYI, D. EMERICK, and OVERHULSE, PAUL R. Segmental aorto-iliac and femoral arterial occlusion; treatment by resection and arterial graft replacement. *J.A.M.A.*, Jan. 29, 1955, *157*, 426–433.

The authors report from the Department of Surgery of the Henry Ford Hospital on the subject of segmental arterial occlusion and the results of surgical treatment. Their reported material includes 27 cases, 19 of which were "pure" segmental occlusion and 8 cases of atherosclerotic segmental occlusion. The most common sites of involvement were in the superficial femoral artery, the bifurcation of the aorta and the popliteal arteries. The authors point out the insidious nature of the disease and the symptoms which are suggestive. The latter are muscular pain brought on by exercise and some peripheral ischemia. The value of angiography is emphasized and typical angiographic findings are illustrated.

The treatment which has been utilized by the authors consists briefly of resection and replacement grafting. They obtained functionally patent grafts and relief of symptoms in 76 per cent of cases.—*C. L. Hinkel, M.D.*

PADEN, PAUL A., and MATZ, FRANK A., JR. Benign systemic hemangiomatosis without cutaneous involvement; a confusing diagnostic problem. *Radiology*, Feb., 1955, *64*, 252–257.

The literature relative to hemangiomas of bone and other organs with associated cutaneous manifestations is reviewed. The authors report a case of an asymptomatic male who had a large dense and expanding hemangioma involving the right parietal bone of the skull and a second similar lesion involving the left sacrum. The spleen was also greatly enlarged. No cutaneous manifestations were present in contradistinction to what was generally reported in widespread bone involvement in the literature.

The authors believe this case represents two simultaneously arising benign lesions. It is difficult to determine whether these lesions should be classified as congenital malformations or spontaneous new growths.—*E. E. Seedorf, M.D.*

DRUCKER, VICTOR, and RODRIGUEZ, CARLOS E. Extensive bilateral calcification within adrenal hemorrhage. *Radiology*, Feb., 1955, *64*, 258–261.

Adrenal hemorrhage is a not infrequent occurrence in infancy and childhood. The hemorrhage may involve one or both glands, may be insignificant, with no destruction of the parenchyma, or may be so extensive as to turn the whole adrenal into a blood sac. Although calcification frequently occurs in localized hemorrhage in other portions of the body, it is rarely found within hemorrhage of the adrenal gland.

The authors report a case which showed an unusual degree of calcification in the adrenal glands. This white female infant lived almost two years after the initial extensive hemorrhage. The calcified deposits were seen in the roentgenograms of the chest, which included the upper abdomen. Intravenous urograms definitely ruled out the kidneys as the site of the calcification. Postmortem examination showed calcification throughout both adrenal glands.—*A. W. Sommer, M.D.*